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THE purpose of the veterinary service is to prevent and cure diseases of animals; its obligation to the nation and to animals themselves is to do so with all the precision contemporary knowledge permits.

Its aim is to develop a profession of college-trained specialists and to maintain the integrity of their occupation.

Its program comprises the conservation of a basal resource, the humane treatment of domestic animals, and coöperation in the preservation of public health.

To attain the objectives of a veterinary service of the higher type, such as the people of the United States require, the unanimous effort of all veterinarians seems in order.

JOURNAL
of the
American Veterinary Medical Association
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(Original Official Organ U. S. Vet. Med. Assn.)

H. Preston Hoskins, Secretary-Editor, 221 N. LaSalle St., Chicago, Ill.

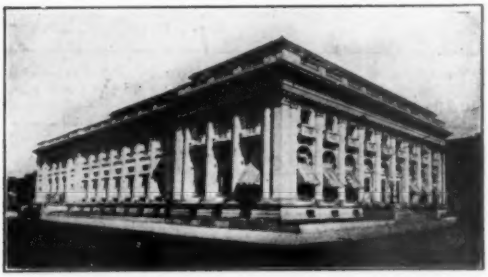
Vol. LXXXVI, N. S. Vol. 39 **JUNE, 1935** **No. 6**

COMMENCEMENTS

Veterinary colleges in the United States and Canada this year will graduate a larger number of veterinarians than for any year since 1920, when the total number of graduates reached 375. This number may be equalled at the 1935 commencements, but it is doubtful. The colleges reported 380 seniors last fall, but all of these are not expected to be graduated this year, for one reason or another. The total number of graduates for 1934 was 299, the largest number in fourteen years. The average number of graduates for the ten years from 1925 to 1934, inclusive, was 187 per year. Next year and in 1937, the number of graduates will be less than for 1935, based on the number of students in the junior and sophomore classes this year. Colleges going to the five-year curriculum account for the expected drop in the number of graduates for 1936 and 1937. We can expect the number of graduates to increase again in 1938 if we take into account the 572 freshmen enrolled this year. In studying these figures keep in mind the estimate made by the A. V. M. A. Committee on Education, several years ago, that 296 new veterinarians would be required each year until 1980 to maintain the numerical strength of the veterinary profession at its 1930 level. Another factor that should not be overlooked is this: All figures on graduates include the two Canadian colleges, while the calculations on veterinarians needed do not include Canada.

NEW MEXICO JOINS THE LIST

New Mexico has joined the list of state associations now affiliated with the A. V. M. A. The decision to affiliate and to have a delegate in the A. V. M. A. House of Representatives at Oklahoma City in August was made at a meeting of the New Mexico Veterinary Medical Association, at Albuquerque, May 4, 1935, according to information received from Dr. T. I. Means, newly elected secretary of the Association. With New Mexico in the fold, there remain only two state associations that are as yet unaffiliated: Wyoming and Arkansas.



FEDERAL BUILDING, OKLAHOMA CITY

COMBINE THE CONVENTION WITH YOUR VACATION

Those from the East, South and Midwest who plan to attend the A. V. M. A. convention in Oklahoma City, in August, will want to plan to see as much of the West as possible while they are convenient to points of interest. What they see will depend, of course, upon the amount of time they can allow themselves away from home. The Grand Canyon in northern Arizona should be visited, if that is at all possible. The Grand Canyon has been called the eighth wonder of the world, and has been described by Joaquin Miller as a 15-by-218-mile paint pot, "the grandest, sublimest thing the world has ever seen."

For those who can not plan such a long trip, the Ozark Mountains in southern Missouri, northwestern Arkansas and a portion of northeastern Oklahoma; and the Ouchita Mountains, which stretch 200 miles from within Oklahoma to central Arkansas, near Little Rock, offer a pleasant vacation stop-over. Hot Springs National Park, in Arkansas, promises scenic wonders that are well worth exploring.

In Oklahoma itself, there are the Kiamichi Mountains, the Arbuckle Mountain and Platt National Park, the Wichita Na-

tional Forest and Fort Sill Military Reservation, the Great Salt Plains and the Claremore Medicinal Springs, any one, or all, of which may be included in a vacation itinerary with profit and pleasure. If it is true that "travel broadens one," then the trip to Oklahoma City should be a broadening and delightful experience.

EXECUTIVE BOARD ELECTIONS

Executive Board elections are now in progress in three districts. Two of these are regular elections to select members of the Executive Board for regular five-year terms. The other is a special election and is being held to fill the vacancy caused by the death of Dr. C. A. Cary. In Districts 5 and 7, where regular elections are being held, the primaries are over and the members are now balloting on the nominees selected in the primary elections. District 5 consists of Iowa and Minnesota, and District 7 consists of Alaska, Hawaii, Idaho, Montana, Nebraska, North Dakota, Oregon, Philippine Islands, South Dakota, Washington and Wyoming. These elections will be brought to a close, June 27.

In District 4 (Alabama, Cuba, District of Columbia, Florida, Georgia, Kentucky, Maryland, Mississippi, North Carolina, South America, South Carolina, Tennessee, Virginia, West Indies and West Virginia), the primary election is now in progress for the purpose of selecting five nominees who will be voted upon later. The polls for this election will remain open until June 24.

President MacKellar has appointed Dr. W. W. Dimock, of Lexington, Ky., as member of the Executive Board to represent District 4 until the successor to Dr. C. A. Cary is determined in the election now being held in the District.

STUDENT CHAPTER AT CORNELL

A student chapter of the A. V. M. A. has been organized at the New York State Veterinary College at Cornell University with 133 members. With the formation of this chapter, the family is now complete. We now have a student chapter in each of the eleven veterinary colleges in the United States and Canada recognized by the A. V. M. A. The first of these chapters was organized at Michigan State College in 1927. During the college year which is just coming to a close, there were 1,042 junior members enrolled in the eleven chapters.

CONGRESS PROCEEDINGS

All of the material constituting the Proceedings of the Twelfth International Veterinary Congress has been in the hands of the printer for some time, and the immense task of reading proof is now in progress. We had hoped to be able to announce at this time a definite date when the Proceedings would be ready for distribution. However, this does not appear to be feasible, and it may be necessary for the Organizing Committee to take the entire twelve months allowed by the statutes of the International Veterinary Congresses for getting out the Proceedings. This would mean that they will not be ready for distribution until some time in August. Just as soon as it is possible to do so, the date of distribution will be announced in the JOURNAL.

KANSAS DECLARED TUBERCULOSIS-FREE STATE

May 1, 1935, was a memorable day for Kansas. On that date, the state was recognized officially by the U. S. Department of Agriculture as the 19th state practically free of bovine tuberculosis and placed on the list of modified accredited areas along with 18 other states: North Carolina, Maine, Michigan, Indiana, Wisconsin, Ohio, Idaho, North Dakota, Nevada, New Hampshire, Utah, Kentucky, West Virginia, Washington, Illinois, Oregon, Virginia and Minnesota.

The testing of cattle for tuberculosis has been speeded up in Kansas during recent months through the use of emergency funds provided by the Jones-Connally Act. This work resulted in the rapid completion of testing in 31 counties which enabled the state to achieve its tuberculosis-free status. Officials who directed the work have commented on the splendid spirit of cooperation on the part of cattle-owners throughout the state.

The campaign to eradicate bovine tuberculosis is progressing rapidly in other states and, during March, 1935, 2,690,074 cattle in approximately 250,000 herds were tested—more than in any previous month in the history of the work.

Two-thirds of all the counties in the United States are now practically free of bovine tuberculosis, according to a map recently issued by the U. S. Department of Agriculture. Of the 3,071 counties in the United States, 2,035 were accredited as of March 1, 1935. In several states, only a few non-accredited counties stand between them and accreditation.

MEMBERSHIP CAMPAIGN

This month, 34 applications for membership are being given first listing. This number of June listings has been exceeded only once in the eleven years during which the present system of handling applications has been in effect. Furthermore, the number of applications listed this month has been exceeded only three times in any month since September, 1929, and two of these occasions were in September when applications filed at an annual convention were given first listing.

Last month, 25 applications were listed, these representing the largest number listed in the month of May for any year since 1929, in which year 26 applications were given first listing. Combining the totals for this month and last, we have 59 applications, and 33 of these have been credited to Oklahoma. The names of three prominent Oklahoma veterinarians have appeared as vouchers very consistently on these applications. A check-up reveals that Dr. C. C. Hisel, State Veterinarian, has signed 22, Dr. L. J. Allen, B. A. I. Inspector-in-Charge, has signed 21, and the name of Dr. C. H. Fauks, A. V. M. A. Resident Secretary, appears on 19 applications.

APPLICATIONS FOR MEMBERSHIP

(See January, 1935, JOURNAL)

FIRST LISTING

BEAUDETTE, FRED R. N. J. Agr. Exp. Sta., New Brunswick, N. J.
D. V. M., Kansas State College, 1919
Vouchers: James J. Black and H. Preston Hoskins.

BRIDGES, BUFORD F. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1934
Vouchers: C. C. Hisel and L. J. Allen.

BRUNKE, ALLEN V. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1933
Vouchers: C. C. Hisel and L. J. Allen.

CLAVELL, CESAR Box 486, Ponce, Puerto Rico.
B. S., University of Missouri, 1933
D. V. M., Texas A. and M. College, 1935
Vouchers: Mark Francis and A. A. Lenert.

COOK, VICTOR J. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1930
Vouchers: T. F. Dunham and Thomas O'Reilly.

CORCORAN, JAMES B. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Colorado Agricultural College, 1934
Vouchers: C. H. Fauks and Thomas O'Reilly.

CUNNINGHAM, GEORGE A. 319 F Ave., Lawton, Okla.
V. S., Ontario Veterinary College, 1904
Vouchers: C. C. Hisel and L. J. Allen.

CURRY, RAY 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1933
Vouchers: C. H. Fauks and C. C. Hisel.

DAVIS, ENNIS A. 1006 13th St., Columbus, Ga.
D. V. M., Alabama Polytechnic Institute, 1922
Vouchers: Don B. Strickler and F. D. Patterson, Jr.

DAVIS, MACK B. 116 N. Harrison St., Shawnee, Okla.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: C. C. Hisel and L. J. Allen.

HATCHER, WILLIAM L. Cimarron, N. M.
D. V. M., Colorado Agricultural College, 1933
Vouchers: S. W. Wiest and T. I. Means.

HEATH, MCKENZIE 1520 2nd Ave. North, Birmingham, Ala.
D. V. M., Alabama Polytechnic Institute, 1919
Vouchers: Louis E. Beckham and C. C. Middleton.

HERBOTT, WALTER K.
Admiral Apts., 48th and Locust Sts., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1916
Vouchers: Thos. Castor and S. K. Nelson.

HOFFMAN, HENRY A. R. 1, Box 326, Petaluma, Calif.
D. V. M., Kansas State College, 1917
Vouchers: E. E. Jones and W. L. Curtis.

HOLLIDAY, JAMES M. Blackwell, Okla.
D. V. M., Saint Joseph Veterinary College, 1923
Vouchers: C. C. Hisel and C. H. Fauks.

HURD, JOHN M. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1934
Vouchers: S. A. Watters and C. H. Fauks.

KETCHAM, HAROLD F. Box 537, Pawhuska, Okla.
D. V. S., Grand Rapids Veterinary College, 1909
Vouchers: C. H. Fauks and L. J. Allen.

LUNSTRA, MITCHELL 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1933
Vouchers: C. H. Fauks and L. J. Allen.

MCCOY, FRANK Manchester, Okla.
M. D. C., Chicago Veterinary College, 1896
Vouchers: C. C. Hisel and L. J. Allen.

MAGATAGAN, VERNON H. Apt. 4, Plunkett, Albuquerque, N. M.
D. V. M., Colorado Agricultural College, 1932
Vouchers: F. L. Schneider and W. L. Black.

MAGINNIS, ERNEST V. Kingfisher, Okla.
D. V. M., Cornell University, 1930
Vouchers: C. C. Hisel and L. J. Allen.

MAXEY, HOWARD C. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Iowa State College, 1933
Vouchers: C. H. Fauks and L. J. Allen.

METCALFE, ELMER L. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas State College, 1934
Vouchers: O. E. Robinson and C. C. Hisel.

MOORE, ROBERT G. Dunlap, Iowa.
D. V. M., Iowa State College, 1913
Vouchers: C. J. Scott and H. Preston Hoskins.

NETHERTON, C. O. 334 Federal Bldg., Oklahoma City, Okla.
M. D. C., Chicago Veterinary College, 1894
Vouchers: G. G. Graham and C. H. Fauks.

PLUMMER, WILBERT A. Laverne, Okla.
D. V. M., Saint Joseph Veterinary College, 1918
Vouchers: C. C. Hisel and L. J. Allen.

SIRILO, LT. ANDREW J. Fort Sill, Okla.
D. V. M., Ohio State University, 1934
Vouchers: C. C. Hisel and L. J. Allen.

SLANKARD, JESSE J. Elk City, Okla.
D. V. M., Saint Joseph Veterinary College, 1921
Vouchers: L. J. Allen and C. C. Hisel.

STORZ, FRED 3200 Muncie Blvd., Kansas City, Kan.
D. V. M., Kansas State College, 1932
Vouchers: C. C. Hisel and L. J. Allen.

TAYLOR, LT. CLARENCE L. Fort Hoyle, Md.
D. V. M., Iowa State College, 1931
Vouchers: Maj. Charles M. Cowherd and Maj. S. C. Dildine.

WATSON, MAJ. HARRY L. Fort Sill, Okla.
D. V. M., Grand Rapids Veterinary College, 1916
Vouchers: C. C. Hisel and L. J. Allen.

WHITCOMB, RUSSELL E. 52 Miller St., Belfast, Me.
D. V. M., Georgia State College of Agriculture, 1931
Vouchers: W. C. Dendinger and J. F. Witter.

WORSHAM, IVEL C. Bixby, Okla.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: L. J. Allen and C. C. Hisel.

WRIGHT, BENJAMIN W. 334 Federal Bldg., Oklahoma City, Okla.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: Roy T. Fisher and C. H. Fauks.

Applications Pending

SECOND LISTING

(See May, 1935, JOURNAL)

Berens, Robert, 9247 Queens Blvd., Elmhurst, Long Island, N. Y.
Bonaci, Alexander J., 2420 Fourth Ave., Seattle, Wash.
Boyce, Lt. Robert A., Jr., c/o Philippine Department, Manila, P. I.
Burgett, Marion V., 1122 W. 143rd St., East Chicago, Ind.
Christy, William L., Tonkawa, Okla.
Coane, Samuel, 1841 Greenwood Ave., Trenton, N. J.
Dolan, Richard L., Jr., 86 E. Church St., Uniontown, Pa.
Enge, Emery H., Comfrey, Minn.
Gump, R. H., 334 Federal Bldg., Oklahoma City, Okla.
Hopkins, David, 21 Laurel St., Brattleboro, Vt.
Hopson, George H., Franklin Ave., Millbrook, N. Y.
Ivey, William E., 334 Federal Bldg., Oklahoma City, Okla.
Jackson, Fay F., Clear Lake, S. Dak.
Kleinfeld, Abraham H., 411 36th St., Union City, N. J.
McGee, George M., 334 Federal Bldg., Oklahoma City, Okla.
McGee, Lucius E., 334 Federal Bldg., Oklahoma City, Okla.
Mitten, James C., 313 Fifth Ave., N. W., Puyallup, Wash.
Morrison, Harold L., 334 Federal Bldg., Oklahoma City, Okla.
Nordstrom, E. Harold, 334 Federal Bldg., Oklahoma City, Okla.
Oelrich, Ivan A., Chancellor, S. Dak.
Peterson, Elliott, 529 W. Canadian St., Vinita, Okla.
Reaugh, George T., 1650 Pattie Ave., Wichita, Kan.
Smith, Roy C., 421 S. Van Buren St., Enid, Okla.
Weaver, Clark A., Bellville, Ohio.
Willers, Karl H., 1028 Greenwood Ave., Canon City, Colo.

The amount which should accompany an application filed this month is $7.91 which covers membership fee and dues to January 1, 1936, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Northeast Kansas Veterinary Medical Society. Jayhawk Hotel, Topeka, Kan. June 4, 1935. Dr. E. H. Lenheim, Secretary, 326 City Bldg., Topeka, Kan.

Texas, State Veterinary Medical Association of, and A. & M. College of Texas Short Course for Veterinarians. Francis Hall, A. & M. College of Texas, College Station, Texas. June 3-5, 1935. Dr. D. Pearce, Secretary, Box 335, Leonard, Texas.

Central New York Veterinary Medical Association. Constableville, N. Y. June 5, 1935. Dr. W. B. Switzer, Secretary, R. 5, Oswego, N. Y.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. June 5, 1935. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

East Tennessee Veterinary Medical Society. Knoxville, Tenn. June 8, 1935. Dr. Robert L. Hummer, Secretary, 312 W. Church Ave., Knoxville, Tenn.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Nebr. June 10, 1935. Dr. J. N. McIlnay, Secretary, 3251 Leavenworth St., Omaha, Nebr.

American Association of Medical Milk Commissions. Joint meeting with Certified Milk Producers' Association. Chalfonte Haddon Hall Hotel, Atlantic City, N. J. June 10-11, 1935. Dr. Harris Moak, Secretary, 360 Park Place, Brooklyn, N. Y.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. June 11, 1935. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. June 12, 1935. Dr. A. S. Schlingman, Secretary, Parke, Davis & Co., Detroit, Mich.

Vermont Veterinary Medical Association. Hotel Coolidge, White River Junction, Vt. July 12-13, 1935. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

New York State Veterinary Medical Society. Binghamton, N. Y. June 13-14, 1935. Dr. F. F. Fehr, Secretary, 243 S. Elmwood Ave., Buffalo, N. Y.

Northeastern Indiana Veterinary Medical Association. Tri-Lake, Ind. June 16, 1935. Dr. H. O. Elliott, Secretary, Box 178, Orland, Ind.

California State Veterinary Medical Association. San Diego, Calif. June 17-19, 1935. Dr. Cliff D. Carpenter, Secretary, 337 Central Ave., Los Angeles, Calif.

Eastern Iowa Veterinary Association, Inc. Mechanicsville, Iowa. June 18, 1935. Dr. John J. Strandberg, Secretary, 1005—8th Ave., Belle Plaine, Iowa.

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. June 18, 1935. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Fargo, N. Dak. June 20-21, 1935. Dr. Lee M. Roderick, Secretary, North Dakota Agricultural College, State College Station, Fargo, N. Dak.

Indiana-Illinois Veterinary Medical Association. Deming Park, Terre Haute, Ind. June 21, 1935. Dr. C. T. Howard, Secretary, Carlisle, Ind.

Northwestern Ohio Veterinary Medical Association. Port Clinton, Ohio. June 26, 1935. Dr. Warren P. S. Hall, Secretary, Division of Health, 9 Ontario St., Toledo, Ohio.

Nebraska State Veterinary Medical Association. (Second annual clinic.) David City, Nebr. June 27, 1935. Dr. E. C. Jones, Secretary, c/o Norden Laboratories, Grand Island, Nebr.

Northwest Veterinary Medical Association. (Joint meeting of the Oregon, Washington and British Columbia Veterinary Medical Associations.) Victoria, B. C. July 8-10, 1935. Dr. W. Graham Gillam, Secretary, Cloverdale, B. C.

Wisconsin Veterinary Medical Association. Chula Vista Resort, Wisconsin Dells, Wis. July 9-10, 1935. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Maine Veterinary Medical Association. Bar Harbor, Maine. July 10, 1935. Dr. R. E. Libby, Secretary, Richmond, Maine.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 10-11, 1935. Dr. E. A. Caslick, Secretary, Paris, Ky.

Virginia State Veterinary Medical Association. (Joint meeting with the District of Columbia, Maryland, North Carolina and West Virginia Veterinary Medical Associations.) Richmond, Va. July 10-12, 1935. Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 11-12, 1935. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Veterinary Medical Association of New Jersey. Hotel Claridge, Atlantic City, N. J. July 11-12, 1935. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Missouri Veterinary Medical Association. Chamber of Commerce Bldg., Saint Joseph, Mo. July 16-17, 1935. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

South Carolina Association of Veterinarians. Claremont Hotel, Sumter, S. C. July 16-17, 1935. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Western Michigan Veterinary Medical Association. Ionia Fair Grounds, Ionia, Mich. July 25, 1935. Dr. C. H. Haasjes, Secretary, 728 S. State St., Shelby, Mich.

Michigan State Veterinary Medical Association. Blaney Park, Blaney, Mich. August 1-2, 1935. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing, Mich.

American Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. August 27-30, 1935. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

STATE BOARD EXAMINATIONS

Iowa Veterinary Medical Examining Board. State Capitol, Des Moines, Iowa. June 11-12, 1935. Further information may be obtained from the Secretary. Dr. H. A. Seidell, Secretary, State Capitol, Des Moines, Iowa.

Nebraska Bureau of Examining Boards. State House, Lincoln, Neb. June 24-25, 1935. Applications must be on file at the Bureau not later than 15 days prior to date of examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Neb.

Massachusetts Board of Registration in Veterinary Medicine. Department of Civil Service and Registration, Boston, Mass. July 9-10, 1935. Applications may be obtained from the Secretary. Dr. E. W. Babson, Secretary, Gloucester, Mass.

Illinois State Board of Veterinary Examiners. Chicago, Ill. July 11-12, 1935. Dr. L. A. Merillat, president of the Examining Committee, asks candidates to file their applications with the Director of Registration and Education, Springfield, Ill., who will notify them of the hour and place of examination.

THE TRANSMISSION OF FOWL LEUKOSIS WITH DESICCATED BLOOD*

By E. L. STUBBS, *Philadelphia, Pa.*
School of Veterinary Medicine, University of Pennsylvania†

Fowl leukosis has been most often transmitted experimentally by the intravenous injection of whole blood from an affected chicken into a healthy one. It has also been transmitted in a variety of other ways, including the use of filtered material. The transmission has required the use of living fowls periodically injected to keep strains of leukosis alive. If material containing the causative agent could be desiccated, it would simplify the routine transfer of this disease and would contribute further information about the nature of the causative agent of leukosis.

A publication by Furth[1] reports an unsuccessful attempt to dry the transmitting agent. An attempt by Jármai,[2] in which he was unsuccessful, indicated to him that the agent transmitting leukosis is different from the agent of Rous sarcoma, that can be dried readily. A later publication by Furth[3] reports further experiments in desiccation that were successful. Those experiments suggested that once the material is successfully dried, the deterioration of the causative agent is very slow. In this report it was shown that the causative agent can be preserved in the dry state but it was thought that the process of drying results in reducing or destroying the transmitting power of the blood.

Experiments were made to investigate the length of time that desiccated material will retain its disease-producing properties.

Forty cubic centimeters of blood was drawn from the jugular vein of chicken 2256 with typical erythroleukosis (strain 1). This was dried from the frozen state *in vacuo* over phosphorus pentoxide, sealed in test-tubes and kept in the refrigerator.

It was estimated that approximately 300 mg of dried blood was obtained from 3 cc of fresh blood. Three cc of distilled water was added to each 300 mg of desiccated blood to restore it to its original volume. This was diluted with physiological salt solution and each chicken tested received an intravenous injection of approximately 60 mg of dried blood.

The chart in figure 1 indicates the groups of chickens injected with this desiccated material and the results of the injections.

First experiment: The first injections were made 40 hours after desiccation. Two of the five injected chickens developed

*Received for publication, December 13, 1934.
†These experiments have been supported by a fund for the study of leukemia.

leukosis, bird 291 showing the first blood changes in 35 days after injection and dying nine days later. The other (292) showed only slight blood changes in 40 days and died five days later. The postmortem examination of these two fowls showed the gross changes typical of leukosis. Microscopic slides prepared from the tissues were characteristic. The three other chickens were kept four months without showing any blood alteration or physical disturbance. At the end of four months they were killed and on postmortem examination showed no evidence of leukosis.

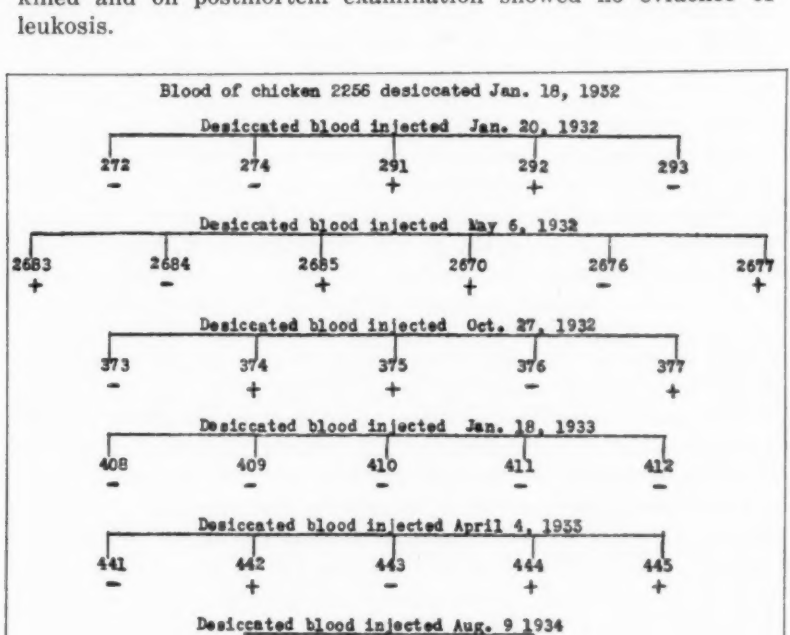


FIG. 1—Chart showing results of injections of desiccated blood.

Second experiment: One hundred and eight days after the blood was dried, six chickens were injected intravenously and four developed leukosis. The first one to develop leukosis (2683) showed the first blood changes 35 days after the injection and was dead three days later. It was our custom to take blood smears weekly and the three other chickens that developed leukosis showed the first blood alteration in the smears taken

on the 46th day after injection. One chicken (2677) was dead three days later, on the 49th day after injection. The second (2685) died on the 53rd day after injection and the third (2670) died the 63rd day after injection.

Third experiment: A little over nine months after the blood had been dried, five chickens were injected and three developed leukosis. The first changes were not noted when the smears were examined 52 days after injection. One leukotic chicken (375) died the following day and at postmortem showed the gross changes characteristic of leukosis. Another (374) died one month later. The third one (377) showed characteristic blood changes for almost one month, after which it recovered.

Fourth experiment: One year after the blood had been dried, five young chickens were injected. None of this group developed leukosis and, when killed four months later, none showed gross changes suggestive of leukosis.

It was felt after the fourth experiment that the dried blood had lost its virulence because it has been unusual not to have some develop leukosis out of five injected with virulent material.

Fifth experiment: One year, two and one-half months after the blood had been dried, a sample was tried that had been kept in a refrigerator other than that in which the dried blood used in the previous tests had been kept. It was injected into five chickens and three came down with leukosis. The first blood changes were noted when smears were examined 21 days after the dried blood was injected. One chicken (442) died 27 days after injection; another (445) died 30 days after injection and a third (444) died 45 days after the dried blood had been injected. Two chickens were badly pecked by other chickens and died too soon for leukosis to be determined definitely on gross examination.

Sixth experiment: Two years, seven and one-half months (932 days) after the blood was dried, the last test was made. This sample had been kept in the same refrigerator as all other samples except that used in the fifth experiment. Three chickens were injected and one developed leukosis. The first blood changes were detected in chicken 306, 46 days after injection. This chicken died 15 days later, when it showed, at postmortem examination, hemorrhages, swollen liver, spleen and hyperplastic bone-marrow. The chicken weighed 690 grams at the time of death. The liver weighed 100 grams and the spleen 9 grams. The changes observed were characteristic of transmissible leukosis.

Discussion

Previous experience indicated that blood of leukotic chickens could be dried and would retain its virulence for a short time. It was thought that some of its disease-producing properties were lost and probably that in some cases it entirely lost its disease-producing ability. Tests were devised to try out such dried material at intervals after drying.

A typical case of leukosis was selected and its blood desiccated. The first test was conducted immediately after the blood was desiccated and resulted in 40 per cent of takes in five chickens injected. It produced the typical changes of transmissible leukosis and was considered satisfactory for such tests. The desiccated blood was kept in sealed test-tubes in the electric refrigerator.

A second test was made 108 days after the blood had been desiccated and resulted in 66 2/3 per cent takes in six chickens injected. Blood changes were produced from 35 to 46 days after injection and caused deaths in all four of the chickens in 38 to 63 days after injection.

A third test was made a little over nine months after the blood had been desiccated and produced 60 per cent takes in five chickens injected. This test produced blood alterations after 52 days and caused deaths in two of the chickens in 53 days and 84 days, respectively, after injection. The third chicken affected in this test recovered.

A fourth test conducted after the blood had been dried one year was entirely negative. It was thought that perhaps the dried blood had lost its virulence at this time but many things can happen in such a test.

Some of the same dried blood, kept in another electric refrigerator, was used for the fifth test one year, two and one-half months after being dried. Sixty per cent of these five chickens came down with leukosis. Blood alterations were produced in 21 days and deaths were produced in 27 to 45 days after the dried blood was injected.

The desiccated blood was tested again 932 days after desiccation and of three chickens injected, one (33 1/3 per cent) came down with leukosis. Blood alteration was produced in 46 days after injection and death occurred 61 days after injection. This shows that the desiccated blood of this chicken, kept in sealed tubes in the refrigerator for 932 days, was still capable of producing leukosis.

SUMMARY

Blood from a chicken with typical erythroleukosis (strain 1) was dried from the frozen state *in vacuo* over phosphorus pentoxide.

The desiccated blood was kept in sealed test-tubes in the refrigerator and tested at various times for longevity of the causative agent.

Tests were made 2, 89, 283, 365, 442, and 932 days after desiccation.

Chickens came down with leukosis in all groups except one.

These experiments indicate that the causative agent of transmissible leukosis of chickens (strain 1) retains its activity in the dry state for as long as 932 days.

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[3]Furth, J.: Studies on the nature of the agent transmitting leucosis to fowls. III. Resistance to desiccation, to glycerin, to freezing and thawing; survival at ice box and refrigerator temperatures. Jour. Exp. Med., lv (1932), pp. 495-504.

New Bulletin on Anthrax

A new bulletin on anthrax has been issued by the U. S. Department of Agriculture. It is known as Farmers' Bulletin 1736-F, Anthrax, and supersedes a former bulletin under the same name. The bulletin may be bought for five cents from the Superintendent of Documents, Washington, D. C.

Michigan Veterinarians to Meet in Upper Peninsula

The 1935 annual meeting of the Michigan State Veterinary Medical Association will be held at Blaney Park, Blaney, in the Upper Peninsula, August 1 and 2. Ever since the time of holding the annual meeting was shifted from winter to summer, some 15 years ago, the meetings have been held at Michigan State College, East Lansing, where splendid facilities are available for the meetings, usually the latter part of June, following the close of the college year. This year, both the time and the place are being changed, and it is believed that it will mark the first time that the Michigan organization has met in the Upper Peninsula. August 1 will be given over to an all-day clinic, and the second day will be devoted to a business session, a few papers and sightseeing. Dr. Edward K. Sales, the Secretary, has extended an invitation for all veterinarians to attend the meeting.

EPIZOÖTIC TICK-BORNE TULAREMIA IN SHEEP IN MONTANA*

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During April and May, 1934, a serious loss of sheep was experienced in a band of 1,320 yearling ewes near Ringling, Montana. The affected band was visited April 23 and 24 by Wilkins, and on the latter date by Mr. Fred Stimpert, of the State Hygienic Laboratory at Helena. Because of the unusually heavy tick (*Dermacentor andersoni*) infestation which was found, a tick-caused condition was suspected. The similarity of the observations to those made by Parker and Dade,[1] who first reported tick-borne tularemia in sheep, suggested this disease. This possibility was supported further by the fact that three of four blood samples secured on April 24 were found by Mr. Stimpert to show low-titre agglutinins for *Bacterium tularense*. Further studies were made coöperatively by the Rocky Mountain Laboratory of the United States Public Health Service and the Montana Livestock Sanitary Board.

The band affected was owned by a large ranching concern and, just prior to the outbreak, had been trailed a considerable distance from winter feeding grounds to the home ranch near Ringling.

ENVIRONMENTAL FEATURES

Since tularemia was suspected to be concerned in the epizoötic and since the occurrence of this disease in ticks is so intimately related to the local small-animal population, an effort was made to secure information concerning environmental conditions, particularly with respect to the native fauna.

The Higgins Bros. ranch, on which the outbreak occurred, is composed of several sub-ranches lying for the most part in arid sagebrush plains, surrounded by low foothills. Of the animal life observed, whitetailed jack rabbits and ground squirrels were dominant; some cottontail rabbits and a few woodchucks, wood rats, field mice and coyotes were seen. Coyotes and badgers were scarce but signs indicated their recent presence in considerable abundance.

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Two of the sub-ranches, about four miles apart, are of particular importance in subsequent discussion, *viz.*, the Quail sub-ranch, where the band was being pastured when the initial symptoms of illness appeared, and Old Dorsey sub-ranch, where the affected band was moved later and the majority of the deaths occurred. The first was adjacent to the foothills about six miles east of Ringling; it consisted of sagebrush-flanked, wild-hay meadows, and was especially heavily infested with jack rabbits and other rodent life. Old Dorsey was mostly a sagebrush area, isolated from the foothills and about 9½ miles north of town, with relatively fewer rodents.

Tick and tick-host abundance: The outbreak was associated with an unusual prevalence of wood ticks, *(D. andersoni)* and local residents were agreed that jack rabbits and ground squirrels occurred in greater abundance than had been observed for several years.

On the Quail place, one of the writers counted eleven jack rabbits while standing about ten minutes in one spot, and in not quite two hours 100 ground squirrels were killed by a person who did not move outside a 20-foot circle.

Counts of rodents seen while driving to and from certain locations were kept in order to obtain quantitative data on the occurrence of tick hosts. A few of these records are quoted to give a further conception of the rodent abundance.

> April 26, 8:30 a.m., 9½ miles between Ringling and Old Dorsey, 17 live and 3 dead jack rabbits were counted along road by driver and passenger, traveling 35 miles an hour. April 27, 7 a.m., 25 miles an hour, same 2 observers, same route, 95 ground squirrels and 8 jack rabbits. April 29, 6:45 a.m., 2.9 miles from Higgins ranch house to Ringling, 20 miles an hour, 101 ground squirrels. May 11, 9 a.m., 3.8 miles between another ranch house and town, 30 miles an hour, 61 gophers and 3 dead jack rabbits.

Adult, unfed ticks were swept from vegetation by a method of "flagging" used in field operations at the Hamilton Laboratory; unfed, adult ticks gathered in this way will be referred to hereafter simply as "drag" ticks. By this method of collecting, fairly accurate quantitative estimates of active tick populations in a selected area at a given seasonal period can be made.

Eighty-six drag ticks were taken in 30 minutes in sagebrush bordering a grassy meadow on the Quail sub-ranch, April 27. From the open, grassy area along the edge of the meadow, 31 ticks were recovered in the same length of time. More than 300 ticks were obtained in about 1½ hours from grassy areas around an old corral nearby. Since sheep are notoriously thorough in removing ticks from vegetation along lines of travel,

the occurrence of adult ticks in the numbers observed on a grazed-over area is convincing evidence of a high initial tick population, especially for a sagebrush environment.

Quantitative counts of ticks on sheep were not feasible, but a remarkably heavy infestation was indicated by the quantity of ticks and tick feces in numerous pockets in the wool, especially about the head and shoulders, together with extensive cutaneous hemorrhagic areas on the flesh side of the pelts of skinned-out sheep that had died. Sixty ticks of both sexes in varying states of engorgement were counted about one ear of one sheep.

FIG. 1. Rabbit 28, too weak to remain standing long, and dying probably from loss of blood due to ticks; 153 adult *Dermacentor andersoni* removed from neck and shoulders. Sparseness of hair over latter and a few engorged ticks are shown.

All jack rabbits shot or found dead showed extensive depilation over the neck and shoulders at the site of tick attachment. The reason for this depilation is not known, though the condition is observed frequently. They carried very few immature ticks, but were heavily infested with adults. From one rabbit, which was active and apparently healthy when shot, 153 adult *D. andersoni* were collected. Another jack rabbit (28 in later discussion) found dying yielded 152 adult ticks. The rabbit tick, *Haemaphysalis leporis-palustris*, which is also a natural carrier of tularemia, was practically absent. Immature ticks were scarce also on the ground squirrels and adult *D. andersoni* do not infest

these animals. By May 12, at the time of a second visit to the area, the adult tick population had decreased markedly on both animals and vegetation.

History of Affected Sheep

The band of yearlings involved was lambed in the spring of 1933. In November of that year, it was moved to winter range near Melville, about 35 miles southeast of Ringling. The band left Melville April 1, 1934, and arrived at the Quail sub-ranch April 8. Presence of ticks was first noticed by the herder April 7, about ten miles out of Ringling.

On April 13, a few "gaunt looking" sheep were noticed. On April 16, two were definitely sick and about 30 more the next day. On April 18, so many looked ill that the owners became alarmed, thinking some poisonous plant might be responsible, and moved the band, with the exception of 38 which were too severely affected to travel. Two, possibly four, sheep died that day and by April 21, 17 had succumbed. On April 25, when the writers arrived, 60 head of moribund sheep and some 400 others, obviously ill, were scattered singly or in varying-sized groups over an area a mile square. Though "deticking" operations were begun by the owners, additional prostrated sheep continued to appear until about April 30.

In all, exactly 200 sheep were lost before the outbreak subsided. Necropsies were performed on as many sheep as time and other activities permitted, and samples of tissues and infesting ticks were saved for laboratory use. The last moribund yearling (44 in discussion below) was necropsied May 8. Except for this animal and a few that were still recovering the epizoötic was ended.

It is of interest that a band of ewes (mothers of the affected yearlings) that had not been taken to the winter range but which had been pastured continuously on another part of the Higgins ranch, were not so heavily tick-infested nor did they become ill though exposed to much the same tick conditions as the yearlings. Both these ewes and the affected yearlings were of mixed breed, predominantly Rambouillet, with some Lincoln. The yearlings, however, had noticeably more open wool, which may have predisposed to heavier tick attachment.

Symptomatology and Related Observations

This subject will be considered in more detail in a separate paper but the semeiotic observations may be briefly summarized as follows: slight dejection at first, stiffness in gait, desultory

feeding, tendency to trail behind when band was moving, head held low, respiration rate noticeably increased, some rise in temperature, and diarrhea often in evidence. As the disease progressed, labored breathing was markedly evident and, in the later stages, often was accompanied by grunting, groaning, grinding of teeth; the walk became increasingly stiff, with a hunched, spraddle-legged appearance of the hind quarters. The temperature in some animals was as high as 107° F. In the very severe

FIG. 2. Sheep 42, raised to feet by hand. Shows typical spraddle-legged posture with lowered head and marked diarrhea. Died 24 hours later. Sheep 32 in background. Head clipped and ticks removed. This animal recovered.

cases diarrhea was usually present at this stage, accompanied frequently by a resultant fly-blown condition with quantities of maggots in evidence in the feces-soiled perianal region. Extreme restlessness and complete prostration finally ensued in the more severe cases, death following in a few hours. In a number of

instances, the removal of infesting ticks before prolonged prostration had occurred resulted in complete recovery in a few days.

The prostrate stage of severe cases could easily be confused with tick paralysis. Paralyzed animals, however, when placed on their feet, are incapable of standing unaided and also show slight or no febrile reaction. By contrast, the Ringling sheep, down with tularemia but not yet moribund, were restless and could be roused to move for short distances or raised by hand to stand alone before lying down again; also fever was a marked symptom. Only one sheep of the Ringling band (46) showed symptoms of paralysis. Whether or not its condition was due to tick paralysis is uncertain. When seen on May 8, it was alert and apparently recovering although there was still a lack of coördination of the right legs. Its general condition and appearance were not indicative of an acute tularemia, but its serum, secured that date, agglutinated *Bact. tularense* in a dilution of 1:640. Recovery of the other affected sheep was uneventful and without observed sequelae.

Lung involvement was almost a constant finding at autopsy, but whether due to tularemia or some concurrent condition was not determined. For example, sheep 10 was moribund April 25, with symptoms comparable to the other sick animals but there was also an excessive nasal discharge. It was autopsied the following day and the picture was comparable, in every respect, to the lesions seen in other animals. Tissues were saved for laboratory tests but no evidence suggestive of tularemia was thus secured. Lung involvement in this sheep was no more marked than in other sheep from which *Bact. tularense* was recovered.

A few sheep in addition to sheep 10 showed considerable mucous discharge from the nose. Here again, the responsibility of tularemia is not clear. While sheep 10 above, without demonstrated infection, showed this condition, sheep 43 in the original Quail group, with a similar nasal discharge and comparable symptoms of illness, showed an agglutination titre of 1:640, and infection was demonstrated in the tissues. Necropsy findings were essentially similar to those of sheep 10.

Anemia was a rather constant finding and was probably the result of the rapid loss of a considerable amount of blood from heavy tick infestation. Hemoglobin determinations were made in the field, using the Tallqvist scale. From sheep selected as apparently normal and others in all stages of the disease as judged by activity and appearance, the following Tallqvist readings were obtained: 5 apparently normal sheep, 60 to 80 per

cent; 5 definitely ill sheep, 20, 20, 30, 40, and 70 per cent; 10 very ill or moribund sheep, 20, 20, 30, 50, 50, 50, 50, 50, 70, and 70 per cent; of 15 recovering or recovered sheep, 20, 30, 50, 50, 60, 60, 60, 60, 60, 60, 70, 70, 80, 80, and 80 per cent. In various species of animals an anemia associated with heavy tick infestations has been frequently observed under both field and laboratory conditions and is the present subject of a special study at the Rocky Mountain Laboratory. Excessive loss of blood due to tick-feeding appears to be the most likely explanation. A very striking instance of this was encountered in a jack rabbit (28) found in a helpless condition in the epizoötic area. It was unable to stand and just able to move its head. The Tallqvist

FIG. 3. Sheep affected with tularemia. Sheep in front unable to rise. The sheep in back has just moved a short distance and is ready to lie down again.

reading was 20 per cent, pulse rate 160, respiration rate 96, temperature 102.6° F. Death occurred within an hour. It was found to be infested with 143 adult and nine nymphal *D. andersoni*. Gross lesions, other than pale tissues, were not evident. Thorough tests of the tissues and of the infesting ticks afforded no evidence of an infectious agent and the blood was found negative when tested for agglutinins for *Bact. tularense*. In experimental studies,[2] the feeding of the number of ticks concerned has repeatedly proved fatal to domestic rabbits, and it

appears probable that this jack rabbit died as a result of exsanguination.

Laboratory Studies

Laboratory tests for the recovery of tularemia infection were made with the tissues and ticks collected from diseased sheep; also with tissues and ticks of native rodents. Blood samples for agglutination tests were secured from both sheep and rodents. Some of those from sheep were purposely taken from animals apparently normal. Unfed adult ticks from nature were collected to be tested for the presence of *Bact. tularense*. A few sheep tissues also were saved for histopathological studies. Most of the tissue samples to be used for infectivity tests were stored in a mixture of one-half glycerin and one-half Sorenson's phosphate buffer solution, pH 7.4. A few others were transported in pure glycerin. Protocols of these tests are found in table I.

Tests of sheep tissues: *Bact. tularense* in pure culture was recovered from the blood of guinea pigs injected with tissues of five of the six sheep from which samples were tested. Tests of external glands (usually the prescapulars) were positive four times and negative twice, of spleen and of liver each positive once and negative three times, of muscle and ulcerous skin negative in one test each. Two attempts failed to recover infection from definitely sick sheep by direct blood transfer to guinea pigs in the field, although agglutination tests made with the same blood samples were positive. The variation in recovery of infection from different tissues may be shown by citation of results of tests of three sheep: (12) spleen, post pharyngeal, prescapular and precrural glands positive, liver negative; (41) parotid, right and left prescapular glands positive, precrural and inguinal glands and spleen negative; (44) liver positive, pooled external lymph-glands, muscle and blood negative. Agglutination titres of 1:1,280 for *Bact. tularense* were observed for serums of the last two sheep but the blood of sheep 12 was not tested.

Tissue samples from three yearlings that died or were moribund when killed and necropsied were submitted to Surgeon R. D. Lillie, of the National Institute of Health, for histopathological study. The details of his report are reserved for use in another paper. His diagnoses, however, were as follows:

> Sheep 12, "subacute pyogenic lymphadenitis, subacute cholecystitis and focal hepatitis"; sheep 30, "the process is not inconsistent with subacute tularemia"; sheep 44, "subacute tularemia lesions in lymph-gland and lung, pyogenic bronchopneumonia,

TABLE I—*Ringling epizoötic, 1934. Test protocols of normal, ill, very ill, moribund, recovering and recovered sheep.*

Sheep	Temp.	Hemoglobin Estimate (%)	Agglutination Titre		Test for Bacterium Tularense in:				
			Bact. Tularense	Br. Abortus	Ticks	Lymph-Glands	Spleen	Liver	Blood
Apparently Normal Sheep									
20	102.8	70	Neg.	Neg.	−				
21	105.2	80	1:160	1:160	−				
22	102.5	60	Neg.	Neg.	−				
Ill Sheep									
2			1:320	1:80					
3			1:160	1:40					
4			1:320	Neg.					
16	104.8								
18	104.4	40	1:160	1:160	+				
19	105.0	70	1:80	1:80					
24	104.0	30	1:160	1:160	−				
25	104.7	20	1:320	1:320	−				
26	106.5	20	Neg.	Neg.	+				
Very Ill Sheep									
11	104.1	50	1:160						
14	103.0	70	1:640	1:160					
15	104.9								
31	105.0	50	1:320	1:320	+				
34	107.0	30	1:1280	1:640	+				
Moribund Sheep									
1			Neg.	Neg.					
10	105.8	70				−	−	−	
12	104.9	50				+	+	−	
13	105.6								
30						+	−	−	
41	104.4	50	1:1280	1:1280	+	+	−		
42	104.4	20	1:640	1:640	+				
43	106.7	20	1:640	1:640		+			
44		50	1:1280	1:1280	−	−		+	−
Recovering Sheep									
32	104.5	50	1:320	1:80	+				
33	105.2	60	1:1280	1:1280	+				
40	104.3	20	Neg.	Neg.	+				
46	103.6	80	1:640	1:320	+				
48	105.4	50	1:320	1:40	+				−
49		60	1:2560	1:1280					
51	104.0	80	1:640	1:320					
52	104.0	60							
Recovered Sheep									
35	103.8	30	Neg.	Neg.	−				
36	102.7	60	1:320	1:320	−				
37	103.3	60	Neg.	Neg.	−				
38	103.2	60	1:640	1:640	+				
39	103.4	70	Neg.	Neg.	−				
47	104.2	70	1:2560	1:320					
50	103.6	80	1:1280	1:320					

midzonal hemorrhagic necrosis of liver, chronic ulcerative cholecystitis."

In tests made at the Rocky Mountain Laboratory, typical tularemia infection occurred in guinea pigs injected with tissues of each of these sheep.

Tests of ticks from sheep: Inoculations of guinea pigs with six of nine groups of ticks collected from sick sheep and from six of ten lots from sheep recorded as recovering or recovered resulted positively. Ticks from five animals selected as normal were negative. In cases of heavy infestation only partial lots were tested.

Agglutination tests: The sera of 36 yearlings of the affected band were tested for agglutinins for *Bact. tularense.* The data are summarized in table II. Twenty-seven of the sera were positive, complete agglutination being caused by serum dilutions varying from 1:80 to 1:2,560; nine were negative.

TABLE II—*Ringling epizoötic, 1934. Agglutination of Bacterium tularense by sera of apparently normal, definitely ill, very ill, moribund, recovering and recovered sheep in the affected band.*

Condition of Animals as Observed in the Field	Animals	Results of Tests		Maximum Agglutination Titre					
		Neg.	Pos.	1:80	1:160	1:320	1:640	1:1280	1:2560
Apparently normal	4	3	1		1				
Ill	8	1	7	1	3	3			
Very ill	4	0	4		1	1	1	1	
Moribund	5	1	4				2	2	
Recovering	8	1	7			2	3	1	1
Recovered	7	3	4			1	1	1	1
Totals	36	9	27	1	5	7	7	5	2

Four sera were from sheep selected as apparently normal; of these three were negative and one positive in a dilution of 1:160. The animal giving the positive test may have been experiencing, or recently recovered from, a relatively mild infection.

Fifteen others were from animals supposed to be recovering or recovered. Of eight from "recovering" sheep, seven were positive in dilutions of 1:320 to 1:2,560. Of seven from "recovered" sheep, four were positive in dilutions of from 1:320 to 1:2,560 and three were negative. As above noted, the group of recovering and recovered sheep had been segregated by the owners on the Quail sub-ranch. The negative tests of recovered

animals may have been due to the inclusion of normal animals among those considered as recovered, or the sheep concerned may have recovered from some condition other than tularemia.

The remainder of the sera, 17 in number, were from sheep definitely ill, and included animals varying from slightly ill to those in a dying condition. Of these sera, 15 were positive and two negative. Agglutinin titres ranged from 1:80 to 1:1,280. The negative tests could mean either that the sheep concerned had not been ill sufficiently long for agglutinins to be present or that they were not ill of tularemia.

The above data show that of 32 tests of ill, recovering, or recovered sheep, 26 were positive and, in conjunction with results of the tests of tissues and of infesting ticks, leave no doubt that the band was affected with tularemia in epizoötic proportion.

All serum samples were tested also for agglutinins for *Brucella abortus* and most of the tests showed a high degree of cross-agglutination (see table III). This phenomenon is encountered frequently with human and other tularemia sera, but such correlative high-titre cross-agglutination was somewhat surprising. The nine sera negative for *Bact. tularense* were likewise negative for *Br. abortus*. Of the 27 positive sera, 13 were of the same titre for both antigens and 13 were of slightly higher titre for *Bact. tularense;* only one serum was completely negative for *Br. abortus;* its tularense titre was 1:320.

TABLE III—*Ringling epizoötic, 1934. Cross-agglutination of Bacterium tularense and Brucella abortus by sheep sera.*

BACTERIUM TULARENSE		BRUCELLA ABORTUS							
ANIMALS	TITRE	NEG.	1:40	1:80	1:160	1:320	1:640	1:1280	1:2560
9*	Neg.	9							
0	1:40								
1	1:80			1					
5†	1:160		1		4				
7	1:320	1	1	2		3			
7	1:640			1	1	3	2		
5	1:1280					1	1	3	
2	1:2560					1		1	
36	Totals	10	2	4	5	8	3	4	

*Includes three sheep selected as apparently normal.
†Includes one sheep selected as apparently normal.

An agglutinin-absorption test was made of serum from sheep 41, which completely agglutinated both *Bact. tularense* and

Br. abortus in a dilution of 1:1,280. After absorption with a concentrated suspension of *Bact. tularense* antigen, it failed to agglutinate either *Bact. tularense* or *Br. abortus* at a dilution of 1:40 or higher.

Tests of "drag ticks": Unfed *D. andersoni* adults, obtained in flagging sagebrush and low vegetation by the method previously described, were divided into lots of not over ten each and allowed to attach to guinea pigs. In this manner a total of 336 ticks from the Ringling area were fed on 42 animals and 223 of the resultant fed ticks were inoculated later into 34 other animals. The tests of both series were entirely negative. A few animals died without apparent cause but spleen transfers all resulted negatively. The test animals were held for observation for 21 or more days before being sacrificed and necropsied.

Tests of ticks and tissues from animals comprising local fauna: Local residents reported observations of dead or dying jack rabbits, ground squirrels, coyotes and sage grouse during the spring and the disappearance of badgers. In the course of our observations numerous jack rabbits and one ground squirrel* also were found dying or dead. In addition, a considerable number of apparently healthy animals were shot, including many cottontails, jack rabbits, several woodchucks, numerous scavenging crows and a golden eagle. When feasible, tissues, blood samples, or ticks were saved for further laboratory studies.

Dead rodents in the area were noticed to be attacked by crows very soon after death, and carcasses could sometimes be located by the presence of these birds. The freshness of the carcass also could be estimated roughly in many instances by the degree of such attack; the eyes on both sides were first picked out and then the liver and spleen were removed, often without further molestation.

Ticks from 24 jack rabbits, one cottontail, ten ground squirrels and two woodchucks were tested by separate inoculation of each lot into test guinea pigs with negative results. Limited facilities prevented separate testing of tissues of all rabbits shot. Results of inoculation of tissues from 18 jack rabbits, two ground squirrels, three woodchucks and one eagle were all negative. Positive results for *Bact. tularense* were obtained by subculture after separate inoculation of ticks from six jack rabbits and of tissues from two. One of these jack rabbits (17) was found within eight hours after death on the Higgins ranch. It was

*It has been observed on other occasions that ground squirrels usually die in their burrows and are consequently less often seen than are dead jack rabbits.

heavily tick-infested and showed gross, though minute, lesions of tularemia in the spleen and liver upon autopsy. Infection was recovered from the ticks and tissues tested. Other tissue samples were preserved and sent for histological study to Doctor Lillie who commented, "The picture is typical of acute tularemia in jack rabbits." He also studied tissues of one of the above woodchucks and reported: "* * the caseous granulomata of the spleen and liver are consistent with subacute tularemia." However, we failed to obtain positive laboratory tests from this animal.

Blood samples from 15 jack rabbits, 21 ground squirrels, two woodchucks and the eagle gave negative agglutination tests, as noted above.

Two rabbits were shot near Harlowtown, about 55 miles east of Ringling and about 36 miles north of Melville, where the yearlings were wintered. Pooled ticks from these two rabbits were inoculated into guinea pigs and a pure culture of *Bact. tularense* was recovered from heart-blood of the moribund animals.

Virulence of strains isolated: Immediately after isolation, nine of the strains of *Bact. tularense* recovered in the above tests were tested for comparative virulence, according to the method of Davis, Philip and Parker,[3] by the cutaneous inoculation of six guinea pigs and six domestic rabbits with first transfer culture of each strain. This allowed comparison of the average survival periods in the several series of guinea pigs and the correlation of these figures with similar data and recovery data of the rabbits. Results are summarized in table IV.

Of the nine strains tested, three were from sheep tissues, three from ticks off sheep, two from tissues of jack rabbit 17 and one from ticks off the same rabbit.

All the test guinea pigs died. The average survival periods of the three lots inoculated with the strains from sheep tissues were 6.0, 6.3 and 6.6 days. The average survival of those lots inoculated with strains from "sheep" ticks were 6.3 6.6, and 7.1 days. On the other hand, the average survival periods of lots inoculated with two strains from jack rabbit tissues and one strain from "rabbit" ticks were 9.3, 9.8, and 9.3 days, respectively.

Comparable results were experienced in the test rabbits. All 36 rabbits inoculated with strains from ticks or tissues from sheep died with average survival periods of from 5.3 and 6.8 days for the six separate groups of rabbits. Of the 18 test rabbits inoculated with three strains isolated from the wild

TABLE IV—*Ringling epizoötic, 1934. Virulence tests with nine strains of Bacterium tularense.*

BACTERIUM TULARENSE		TEST ANIMALS															
STRAIN	SOURCE	RABBITS INOCULATED	SURVIVAL (DAYS)						AVERAGE	GUINEA PIGS INOCULATED	SURVIVAL (DAYS)						AVERAGE
			R-1	R-2	R-3	R-4	R-5	R-6			GP-1	GP-2	GP-3	GP-4	GP-5	GP-6	
60954	Sheep 30 Prescapular Gland	6	4	4	5*	6*	6	7	5.33	6	6	6	6	6	7	7	6.33
61362	Sheep 41 Prescapular Gland	6	4	5*	6	6	6	6	5.50	6	6	6	6	6	6	6	6.00
60924	Sheep 43 Prescapular Gland	6	5	5	5	5	6	7	5.50	6	6	6	6	6	7	9	6.66
61440	Sheep 34 Ticks	6	4	5	5	6	6	8	5.66	6	5	6*	7	7	8*	†	6.60
61452	Sheep 41 Ticks	6	5	5	5	6	7	7	6.83	6	5	6	6	6	7	8	6.33
61730	Sheep 46 Ticks	6	4	4	6	6	6	7	5.50	6	6	7	7	7	7	9	7.16
60903	Rabbit 17 Spleen Tissue	6	†	+	+	+	+	+		6	8*	9	9	9	10	11	9.33
60906	Rabbit 17 Liver Tissue	6	+	+	+	+	+	−		6	7	9	10	10	11	12	9.83
61458	Rabbit 17 Ticks	6	+	+	+	+	+	−		6	8	9	9	10	10	10	9.33

*First inoculation negative; reinoculated with same strain.
†Test valueless.
+Agglutination test positive on 14th day; animal survived.
−Agglutination test negative on 14th day.

rabbit and its infesting ticks all survived; none appeared ill at any time, with the exception of one animal that was injured and had to be sacrificed. Nearly all test animals showed a local lesion at the site of inoculation. Blood samples were taken from the 17 surviving rabbits on the 14th day following inoculation and, with only two exceptions, were positive in agglutination of *Bact. tularense* at 1:160 or higher dilutions.

Two strains isolated from two additional rabbits were tested after refrigeration of the cultures for two months. They were of even lower virulence than the strains from rabbit 17, cited above. The average survival period for six inoculated guinea pigs was 10.1 days for one strain and 12.3 days for the other. One guinea pig inoculated with the second strain survived till the 17th day and showed extreme, but typical, pathological changes upon necropsy. All twelve rabbits of the two groups inoculated with these two strains survived the infection, and serum samples on the 14th day were positive for *Bact. tularense* at 1:160 or higher.

The above results with guinea pigs and rabbits indicate a definitely greater degree of virulence for the strains isolated from sheep tissues and from ticks from sheep than for those isolated from the jack rabbit and the "rabbit" ticks. According to the classification proposed and used by Davis *et al.*,[3] the strains isolated from "sheep" ticks and sheep tissues could be considered as extremely virulent, and those from the rabbit as relatively mild. Unfortunately, however, the number of strains used and the data obtained are not extensive enough to justify a conclusion to the effect that this difference in the virulence of strains from sheep and rabbit materials would hold consistently in the area studied. Nor can it be said that the virulence of the two refrigerated strains had not been affected by storage.

Disease in Local Residents

Considering the extent of the outbreak of tularemia in the domestic and native animals and the considerable human contact both with animals and ticks, it is remarkable that only one human case occurred in the area to the knowledge of the writers. A child of four years, living on a ranch about five miles from Old Dorsey, had been bitten by many ticks during the spring. One bite on the right side became irritated and refused to heal. About "a weeek or 10 days" after removal of the ticks, the child was confined to bed (April 26). Tularemia was suspected by Dr. R. J. O'Neill, of White Sulphur Springs, and the symptoms were typical when the patient was seen by Philip, May 7. The

right axillary glands became enlarged and there was an erythematous area about a persistent vesicle at the point of bite. Sickness lasted over a month. A blood sample drawn early in May gave a negative agglutination test, while one drawn on May 20 was positive in a dilution of 1:640.

Several employés on the Higgins ranch were constantly engaged, working bare-handed, in skinning out dead sheep, picking ticks and otherwise handling sick sheep and contaminated materials during the outbreak. Abrasions were even noted on some employés' hands which were repeatedly covered with blood of dead sheep. No precautions were taken against infection. The writers exercised such care as was possible under the field conditions, but broken gloves, accidents and other contingencies incident to handling carcasses and ticks constituted excellent opportunity for infection. No suggestive indispositions occurred among any of the Higgins employés, and blood samples of seven, drawn April 30, and of one of the writers, drawn later, showed no agglutinins for *Bact. tularense*.

COMMENT

Bact. tularense was isolated in pure culture from one or more of the tissues from each of five of six dying or recently dead sheep from which tissue specimens were tested, from six of nine groups of ticks collected from sick sheep, and from six of ten lots from sheep recorded as "recovering" or "recovered." Ticks from five normal animals were negative. Also *Bact. tularense* was agglutinated in considerable titre by the sera of 15 of 17 sheep in various degrees of illness, seven of eight that were "recovering" and by four of seven that were "recovered." These data, together with the histopathological evidence of tularemia lesions in the tissues of affected sheep, indicate that tick-borne tularemia infection was prevalent among the band in epizoötic proportions, but the evidence as to whether or not tularemia was the only cause of the 200 deaths is less clear. There is doubt on this point for two reasons: first, that it is uncertain whether the excessive lung involvement noted in known tularemia-infected sheep was due wholly or only in part to *Bact. tularense*, and second, that an anemia, presumably due to rapid loss of blood by tick-feeding, was quite general in ill animals.

Parker and Dade[1] did not report lung involvement in tularemia-infected sheep in Idaho and Dade has recently informed us that he has never encountered such in the course of his experience with epizoötics of tularemia in sheep in that state. It appears, therefore, that lung pathology is not always associated with

tularemia in sheep. In the Ringling epizoötic, however, the evidence suggests that, though part of the lung lesions were due to *Bact. tularense* (see sheep 44), some at least may have been due to some other infectious agent. The latter possibility could have been a contributory cause of death of sheep 10 (discussed previously), for example, as indicated by the negative test data. However, the variables of testing, such as storage, transportation, etc., do not preclude the presence of tularemia infection in this yearling and the necropsy findings strongly suggest that it was infected.

Besides the lung pathology referred to above, three other conditions not recorded by Parker and Dade were observed in affected animals in the Ringling epizoötic, namely, a frequent low carriage of the head (rather than high), grinding of the teeth in animals seriously ill, and marked distention of the gall-bladder which was noted in each animal autopsied.

With respect to the possible part that may have been played by exsanguination anemia due to tick feeding, the Tallqvist data from affected sheep and the prior experimental evidence which has been secured showing that this condition alone can produce death suggest that this also could have been at least a contributory factor in some of the fatal terminations. The relatively rapid recovery of affected sheep when infesting ticks were removed indicates a correlated increase in the ability of the system to combat tularemia. However, it is impossible to be certain that tick removal did not terminate some other process as well as the withdrawal of blood (*e.g.*, continued reinfection with *Bact. tularense*).

In view of the number of tularemia-infected ticks and animals shown to be present by laboratory tests, two of the observations made are somewhat puzzling, *viz.*, (1) the failure to recover infection from any of the locally-collected, unfed or "drag" ticks, and (2) the failure of any exposed persons to become infected in spite of extensive handling of ticks, tick-feces-contaminated wool, and infected tissues.

The development of the epizoötic was almost explosive, some 40 per cent of the band becoming ill in a period of not over 24 days. Duration and intensity of the disease in individual sheep varied; some recovered after dipping and removal to new pastures with only short indisposition, while others, notably many of those left on the Quail place, must have been desperately ill for two weeks before their illnesses terminated in either death or recovery. Information was not obtained concerning the time

and degree of bacteremia in infected sheep, direct blood transfers to guinea pigs having been attempted too late in the epizoötic to obtain significant results.

It appears probable that the infection in the Ringling sheep was picked up after their arrival on or near the Quail sub-ranch from winter range in consideration of (1) the presence of the disease in probable epizoötic proportions among the local rodents, (2) the exceptional abundance of ticks, and (3) a lapse of approximately eight to ten days after arrival before appearance of the first group of definitely sick sheep and the fact that sheep continued to become ill for some days thereafter.

On the other hand, two circumstances suggest the possibility of the infection being imported by the sheep, *viz.*, the complete failure to demonstrate infection in the considerable sample of "drag" ticks from the Quail sub-ranch, and the recovery of infection from ticks off two rabbits (the only ones secured) taken at random not far from the winter range of the sheep near Harlowtown. While the tests of "drag" ticks are puzzling, any negative significance which one might be inclined to attach thereto is minimized by the demonstration of tularemia infection in the jack rabbit population of the Higgins ranch and ticks infesting them. Obviously, infection was present in the local tick fauna in spite of the test results.

The above observations, though adding materially to the information concerning tularemia in sheep, nevertheless emphasize the fact that there is much to learn, and indicate the value of more intensive studies at future opportunities.

SUMMARY

An explosive outbreak of disease occurred among sheep near Ringling, Montana, in April and May, 1934. Of 1,320 yearling ewes, approximately 40 per cent were symptomatically affected and 200 died before the epizoötic subsided. Observations made locally showed (1) unusually heavy infestation of the sheep by wood ticks *(Dermacentor andersoni)*, (2) appearance of illness following removal from winter to summer range with estimated incubation period of eight to ten days, (3) coincident mortality among tick-infested jack rabbits, (4) subsidence of the epizoötic with reduction in the local tick population (although aided by mechanical control of ticks as far as the sheep were concerned) and (5) occurrence of a human case of tick-transmitted tularemia. Tularemia infection was largely responsible for the epizoötic as indicated by the following laboratory data: (1) re-

covery of *Bact. tularense* from tissues of five of six affected sheep and from two of 18 dead or killed whitetail jack rabbits, (2) recovery of that organism from twelve of 19 samples of ticks off infected sheep and from six of 24 samples of ticks off dead or killed jack rabbits, and (3) positive agglutination tests with 26 sheep sera of 32 apparently affected animals in titres roughly corresponding to the stage of illness or of recovery. Only one of four sera from apparently normal sheep showed agglutinins and then in low titre. Histopathological study of tissues of two of three affected sheep and of one rabbit confirmed the positive test data on these animals.

In view of the amount of tularemia infection in ticks and animals shown to be present, two observations are particularly puzzling: (1) the failure to recover *Bact. tularense* from unfed ticks collected locally and (2) the failure of the owners, employés, or investigators to become infected in spite of massive exposure to infected carcasses, ticks and tick-feces-contaminated wool.

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Women Veterinary Students at O. S. U. Organize

Unique among organizations of veterinary students is the club established at Ohio State University by the women students in the College of Veterinary Medicine. The organization, which was completed at a dinner meeting on May 1, 1935, has been named the Brumley Club in honor of Dean O. V. Brumley who has been active in promoting the cause of women students in veterinary medicine. Dr. Brumley was made an honorary member of the club, and Miss Grace Zorbaugh, associate dean of women, was elected faculty adviser. Officers serving the new group are: President, Miss Ernestine Kelly, of Baltimore, Md.; secretary-treasurer, Mrs. Ida Mae Dodge, of Butte, Mont. The roster of charter members includes the following names, in addition to the officers: Misses Helen Zimmermann, Betsy Jane Richey, Elizabeth Newkirk and Lois Hopkins, and Mrs. Barbara Peters. Future meetings of the club will be dinner meetings and will be held the first Wednesday of each month beginning with the next college year.

BANG'S DISEASE IN BISON AND ELK IN THE YELLOWSTONE NATIONAL PARK AND ON THE NATIONAL BISON RANGE*

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It has been known for some years that Bang's disease has occurred in the bison, more commonly known as buffalo, in the Yellowstone National Park and on the National Bison Range in western Montana. Mohler,[1] in 1917, reported positive agglutination reactions in the blood of two buffalo cows which had aborted in the Yellowstone Park herd. In 1930, Creech[2] reported the isolation of *Brucella abortus* from a diseased testicle of a buffalo killed on the National Bison Range. Rush,[3] in 1930, obtained tests on serum samples from five buffalo in the Yellowstone National Park herd, with positive results in three cases.

At this time, W. M. Rush, of the United States Forest Service, who was in charge of wild life studies in the Park, became interested in obtaining further information on the extent of Bang's disease in the buffalo. He requested the assistance of the Montana Veterinary Research Laboratory, which was gladly given, as it was thought that any information obtained would be of general interest in connection with the study of Bang's disease in domestic cattle.

AGGLUTINATION TESTS ON SLAUGHTERED BUFFALO

In December, 1931, 205 buffalo were slaughtered from the Yellowstone Park herd in order to reduce the size of the herd. Agglutination tests for infectious abortion were conducted on the serums of 90 cows, 13 bulls and 3 steers, using the tube test with serum dilutions of 1:25, 1:50 and 1:100. Definite agglutination at 1:50 or 1:100 was considered positive evidence of infection. It was found that a high percentage of the slaughtered animals were positive to the test. Twelve (92 per cent) of the 13 bulls, all of the three steers, and 49 (54 per cent) of the 90 cows reacted positively.

In 1932, blood samples were collected from 199 slaughtered buffalo cows, steers and bulls. The serums were tested by the rapid agglutination test, using a 1:50 dilution. All serums showing more than a trace of agglutination in this dilution were classified as positive. Twenty-five (41 per cent) of 60 steers, 30

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(61 per cent) of 49 bulls, and 52 (58 per cent) of 90 cows reacted.

In 1933, blood samples were obtained from 59 cows and 10 bulls slaughtered. The standard tube and rapid plate agglutination tests were used. The same standard of selecting reactors was again used as in the two preceding years. Nine (90 per cent) of the 10 bulls and 42 (72 per cent) of the 59 cows were classified as reactors.

AGGLUTINATION TESTS ON LIVE BUFFALO IN 1933

The buffalo cows selected for slaughter in 1931, 1932 and 1933 were for the most part the older, off-type animals. It was thought that more reliable information would be obtained as to the actual status of the disease in the herd if a number of young cows could be tested, permanently identified, and retested over a period of years. Blood samples were from 78 young cows and four bulls selected from 550 animals in the corrals. All of the bulls and 55 (70 per cent) of the cows were reactors. Classifying these cows as to ages, 83 per cent of the three-year-olds; 68 per cent of the four-year-olds; 68 per cent of the five-year-olds, and 50 per cent of the six-year-olds were positive. This test shows a marked decline in reactors from three to six years of age. These tests give very definite evidence of the activity of the infection in this herd.

BLOOD TESTS OF BUFFALO FROM NATIONAL BISON RANGE

A number of buffalo were slaughtered in 1932 and 1933, at the National Bison Range, Moiese, Montana, for the purpose of reducing the size of that herd to conform to the range on which they must be held. Blood samples from all the animals slaughtered were tested at the laboratory.

In 1932, tests were made on eight cows and 79 bulls. Four (50 per cent) of the cows and 54 (68 per cent) bulls reacted to the agglutination test. Three of the positive bull serums were titrated, using dilutions up to 1:6,400. One serum agglutinated completely at 1:400, and the two others were complete at 1:1,600 and partial at 1:3,200.

In 1933, blood samples from 20 bulls and 66 cows, butchered at the National Bison Range, were tested by the agglutination test. Twelve (60 per cent) of the bulls and 36 (54 per cent) of the cows reacted to the test.

PERCENTAGE OF CALVES

It is a difficult undertaking to figure the percentage on a calf crop from the buffalo in Yellowstone Park, since their range

covers many square miles of very rough mountainous land in the northeast corner of the park. Only during the most severe winter weather is it possible to obtain a fairly accurate count of the animals, and even then many small bands of buffalo fail to come in to the lower range that is accessible to man. During mild winters such as 1933-34, so few buffalo come out of the high country that even a rough estimate of numbers is impossible.

FIG. 1. Bison in Yellowstone National Park.

A fairly accurate idea of the calf crops of 1931 and 1932 was obtained. These figures are based upon counts through the winter, at which time the calves were six to eight months old. The calf crop of 1931 was accurately estimated at 55 per cent by an actual count of 239 calves and 279 cows, with 20 calves and 190 cows estimated to have been left in the hills. For 1932, the estimate was 69 per cent by a count of 135 calves and 196 cows, with 38 calves and 54 cows estimated in the hills. It is believed the majority of the 1933 calves were counted, numbering 143, although the percentage cannot be given, since the cows were so scattered it was not practical to count them.

Very little information is at hand regarding the calves born from the herd of buffalo at the National Bison Range, although no actual abortions were observed in 1932. One hundred twenty-four living calves were born from 163 of the cows over three

years of age. Some three-year-old cows do have calves, so the percentage there is slightly less than 74 per cent. Evidently there has been very little ill effect upon the reproductive organs of these cows up to the present time, since a 70 per cent calf crop from range cattle free from Bang's disease would be very close to an average for Montana.

Reproductive Organs of Bulls

In addition to the blood test, an examination was made of the reproductive organs of a number of bulls of both the Yellowstone Park and National Bison Range herds. In 1932, 26 pairs of testicles were forwarded to the laboratory for examination. These specimens were not selected. Gross lesions were found in only one testicle, which was from a reacting bull. A pure culture of *Brucella abortus* was recovered from this lesion. Sixteen of the 26 bulls from which the testicles were taken were reactors.

In 1933, the testicles of ten bulls, taken at random, were forwarded to the laboratory. A careful examination for gross lesions was made, with the results shown in table I. In six cases, some abnormality was noticed. In one case (55) there was a definite lesion from which *Brucella abortus* was recovered in pure culture.

Table I—*Examination of buffalo testicles received from Yellowstone National Park herd, 1933.*

Buffalo	Gross Pathological and Bacteriological Examinations	Agglutination Test
33	One testicle atrophied, epididymis large and fatty. The other testicle somewhat shrunken, the tunica vaginalis adherent, the epididymis large and fatty	Negative
37	No lesions	Positive
38	One minute pus focus in tail of epididymis. Smear negative	No test
46	Minute pus foci in tails of both epididymi. Smear negative	Positive
47	No lesions	Negative
53	No lesions	Negative
54	Minute pus focus in tail of one epididymis. Smear negative	Positive
55	Tail of epididymis much enlarged, contained thin milky pus. *Br. abortus* in pure culture	Positive
57	Lesion in head of epididymis, an area where the tubules stand out prominently and apparently contain a little pus. Smear negative. Minute pus foci in tails of both epididymi	Positive
67	No lesions	Positive

At the slaughter of the bulls from the National Bison Range in 1932, particular attention was directed toward the reproductive organs. Six testicles showing gross lesions were forwarded to the laboratory for pathological and bacteriological examination. Proper identification was not made of this group of testicles, so that the blood reaction of the bulls from which the organs were taken is not known. The results of the observations are as follows:

Testicle 1: Testicle proper apparently normal. The body of the epididymis showed thickening and yellowish areas on the surface. On section there were numerous small pus foci in three groups, from which came a smooth, slightly yellowish pus. *Br. abortus* in pure culture was isolated from the epididymis.

Testicle 2: Testicle was very much enlarged, consisting of a mass of creamy, yellowish pus. Direct smears showed nothing definite.

Testicle 3: Testicle proper contained many abscesses composed of a smooth semi-solid pus. Cultures yielded mixed staphylococcus and a small Gram-negative rod resembling *Br. abortus*.

Testicles 4, 5 and 6: Testicle entirely replaced by a mass of pus in the tunica vaginalis.

Test for Bang's Disease Among Yellowstone Park Elk

During the winter of 1931, blood samples were tested from cow elk running in the vicinity of the buffalo ranch. Thirty-two blood samples were subjected to the agglutination test for Bang's disease. On this number, eleven agglutinated in some degree. Six (18 per cent) agglutinated in a 1:50 dilution strongly enough to be classed as reactors, and five others showed a trace in 1:50 or 1:25. These results indicate that the elk running on the buffalo range had been exposed to the buffalo infection, and that the infection had established itself in some of the elk.

In 1932, blood samples were obtained from 66 elk on the buffalo range. Of this group, two bull elk, one young cow and 49 mature cows were negative. One young cow and ten mature cows were suspicious, reacting in the low dilution of 1:25. Three mature cow elk (4 per cent) were positive, two giving complete agglutination at 1:100, and the third complete at 1:50 and partial at 1:100 dilution. Seven blood samples were tested in 1933 from cow elk running on the buffalo range. These serums failed to show any agglutination of the *Br. abortus* antigen.

Another herd of elk is located in the vicinity of Mammoth Hot Springs, some 30 miles distant from the buffalo ranch. These two herds are apparently quite distinctly separate as to their

range. Blood samples were obtained from 36 elk in this herd. Two samples (5 per cent) were classed as positive, although they showed incomplete agglutination at 1:100 dilution, and the 34 others were negative.

Ten blood samples from cow elk were received from the Mammoth herd in 1932. All of this number were negative to the agglutination test.

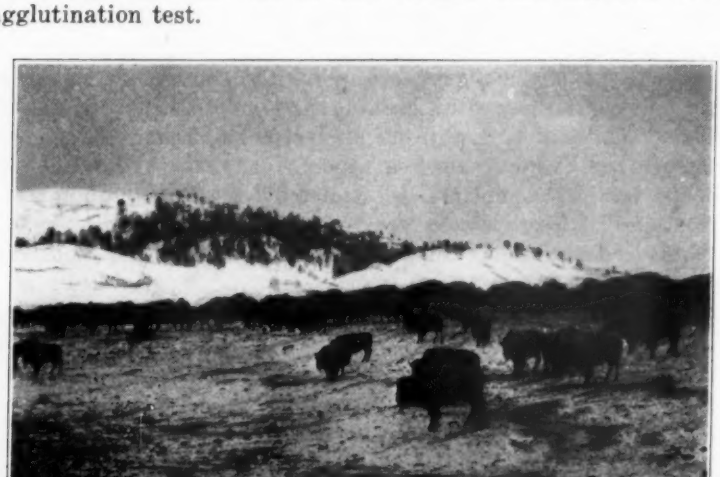

FIG. 2. Another view of a bison herd.

DISCUSSION

The presence of Bang's disease in the Yellowstone Park buffalo was established in 1917. There is no possibility of determining whether this was a disease of the American bison before the introduction of domestic cattle into the buffalo country. In this instance there is a known possible source of infection from domestic cattle. Rush[3] says:

> A few domestic cattle were maintained on a part of the buffalo ranch until about 1919 * * * * this is the only contact the buffalo have had with domestic stock * * * *.

An unusually high percentage of reactors of both sexes was found in this buffalo herd. This was especially true of the steers and bulls. It is particularly remarkable that 41 per cent of 60 steers reacted to the test. Bulls of the domesticated beef and dairy cattle occasionally become infected. Naturally, there are fewer bulls in herds of range cattle than in the buffalo herd,

but the percentage of reactors is never high. This suggests that buffalo may be more susceptible to Bang's disease than are domesticated cattle. Since 70 per cent of young cows tested in 1933 were reactors, it seems safe to conclude the infection is very active in the Yellowstone Park herd. Additional evidence pointing to activity of the infection is found in the results of the testing of serums from slaughtered cows for three years. In 1931, 1932 and 1933, the reactors were 54 per cent, 58 per cent and 72 per cent, respectively, indicating a pronounced increase in the number of positive females in the herd.

As a record of the incidence of abortion is not available, it is impossible to determine just what effect the disease is having upon the reproductive activity of the cows. We have attempted to gain some information on this point from observations on the condition of the reproductive organs at the time of slaughter. While the cows slaughtered each year represent to a large extent the older cows in the herd, the extent of the infection in this class of cows is apparently about the same as in the young cows. In the 1933 test, 72 per cent of the slaughtered cows reacted, as compared with 70 per cent reactors among the living young cows tested.

Notes were made when the cows were slaughtered as to whether their mammary glands were active, indicating that a calf had been raised during the past year, and as to whether the cows were pregnant. On the basis of these observations, an attempt was made to determine whether there was any correlation between the breeding condition of the cows and their reactions to the agglutination test. The result of this study was negative. There was a decrease in the percentage of lactating cows from 1931 to 1933, but this percentage was no smaller in the reacting group than in the non-reacting group.

The only conclusion which can be drawn is that, while the infection is evidently active in the herd, any reproductive inefficiency which may exist is not entirely due to Brucella infection in the cows.

The results of the three years of testing in the Yellowstone herd show 72 per cent of 76 bulls, 44 per cent of 63 steers, and 66 per cent of 301 cows as reactors to the test. The records of the slaughtered buffalo from the National Bison Range for two years show 66 per cent of 99 bulls and 54 per cent of 74 cows as reactors.

The number of calves has been greater than would ordinarily be expected among range cattle with such a high percentage of

reacting cows. The calf crops from the Yellowstone buffalo for 1931 and 1932 were figured at 55 per cent and 69 per cent, respectively. No estimate was made for 1933, although 143 calves were counted, which would probably give a figure between those for the preceding years. At the National Bison Range the calf drop seems to be even higher, about 74 per cent, with 124 living calves born in 1932.

Br. abortus was isolated from pathological testicles of two reacting bulls from the Yellowstone Park herd in 1932 and 1933, and from the testicle of one reacting bull from the National Bison Range in 1933. These cultures were submitted for typing tests to Dr. I. F. Huddleson, who reports that they have all the characteristics attributed to those of bovine origin.

The possibility was suggested that elk grazing over the buffalo range may have picked up the infection. This point was borne out by blood tests conducted on 105 elk serums in 1931 and 1932. Of this number, 8 per cent reacted positively, and 14 per cent gave suspicious reactions in a 1:25 dilution. These reactions would indicate some of the elk on this range had at least become sensitized to the Bang infection. There has been very little mingling of these elk from the buffalo range with a small herd some 30 miles distant, in the vicinity of Mammoth. Blood tests of the latter herd should give some idea of the normal agglutinins carried by elk for *Br. abortus*. During 1931 and 1932, 45 of these elk serums were tested. No positive reactions were found, but 2 (4 per cent) gave suspicious reactions at 1:25. These figures would confirm the suspicion that elk may become sensitized to *Br. abortus*, and the higher number of reactors among elk running on the buffalo range indicates the buffalo as the source of infection.

ACKNOWLEDGMENTS

The authors wish to thank the personnel of the Forest Service and the National Park Service, and especially W. M. Rush, of the Forest Service; George F. Baggley and Francis D. LaNoue, of the Yellowstone National Park Service, and Dr. Robert S. Norton, of the National Bison Range, for the collection of specimens and information that have made this study possible.

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OBSERVATIONS ON THE PATHOLOGY OF BLIND STAGGERS AND ALKALI DISEASE*

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The Third Annual Report issued by the Wyoming Agricultural College and Experiment Station, in 1893, alluded to a peculiar disease of horses in Carbon County, Wyoming. The symptoms noted were loss of flesh, manes and tails partially or wholly depilated, the hoofs elongated and cracked, and the long bones of the body eroded. The causal agent suspected was hay obtained from native grass lands that had been placed under irrigation. The soil upon which the hay grew was of Cretaceous origin, geologically known as Steele shale. At that time, this disease was diagnosed as a form of ergot poisoning. More recent investigations[1] have established the fact that similar symptoms in live stock can be produced from grasses, cereals, and other forages containing the toxic mineral selenium.

Other toxic minerals also may be involved. For example, investigators at the Wyoming Experiment Station have grown barley on experimental plats where the soil was fertilized with sodium molybdate; the barley so obtained was found to be capable, when fed to cattle in small daily amounts, of producing pathological lesions similar in many ways to those caused by feeding barley containing selenium. Since molybdenum has been found to occur in various types of native range plants in this region, it is inferred that molybdenum should be included in the general study of malnutrition of live stock. Tellurium is another element that is being investigated in connection with the general problem of toxic feeds and forages.

In Wyoming the number of reported cases of live stock exhibiting outwardly the symptoms referred to above is not in the aggregate significant enough to be considered except as a minor problem. The name "alkali disease" has been used to designate this type of mineral poisoning.

Various forms of malnutrition and acute plant poisoning in Wyoming, resulting from live stock feeding upon weeds and browse containing selenium and other toxic minerals, present a problem that is much broader in its scope than the so-called "alkali disease." The term "blind staggers" has been used to designate this general type of live stock poisoning by stock-

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men. This term in a technical sense is misleading, because afflicted animals may not become blind, nor do they necessarily stagger about. In fact, in acute cases an animal seldom struggles during the duration of the intoxication. Likewise, the term "alkali disease" is misleading in a technical sense in that the ordinary alkalies are not the primary causal sources of the disease. In view of the fact that no other terminology has been suggested that could satisfy the conditions as they exist and the further fact that these terms are generally accepted and used by stockmen to differentiate what outwardly appear to be two distinct types of toxic mineral poisoning, the authors have chosen to retain the two designations "alkali disease" and "blind staggers" in the discussions that follow.

In a recent publication,[2] the symptoms and pathology observed in live stock suffering from alkali disease and blind staggers, forms of plant poisoning noted in certain areas of Wyoming, were described. It was pointed out that some of the symptoms noted in the affected animals and the lesions observed at autopsy were quite similar and that to designate the two types of injury as distinct and different was not entirely justifiable. On the other hand, the eye lesions noted in a typical case of blind staggers and the tendency of the animal to wander aimlessly, often in circles, are very characteristic of the more advanced cases and are not observed in a true case of alkali disease, so that it is thought desirable to retain the two terms. Many ranchers refer to either type of injury as "alkalied animals," "mineralized animals," or "locoed animals." The symptoms, especially the nervous injury, observed in animals poisoned by the loco plant are different, and to associate locoism with blind staggers cannot be justified.

Briefly summarizing from our previous publication,[2] it is believed that both types of injury are due to the same or related causative agents, namely, small quantities of certain toxic elements or compounds occurring in the vegetation grazed. Serious nutritional disturbances are noted in both types of injury. There is a disturbance of the calcium-phosphorus metabolism. Animals suffer from avitaminosis, especially from vitamin-A deficiency. Anemia and icterus are common to both forms of poisoning. Bone and hoof lesions are constantly observed in alkali disease. However, erosion of the articular surface of the long bones is also rather prevalent in blind staggers.

Animals suffering from either type of injury soon exhibit roughened, off-color coats and assume a rather characteristic stance. Alkali disease cases have long, crooked hoofs and walk

with a peculiar gait, stepping as if they were trying to clear a high object in their path. Animals affected from either disease look prematurely old. The offspring are sometimes degenerate. The authors have observed a few badly affected ranches where it would appear that a definite trend toward stuntiness in cattle has resulted through a cycle of a few generations. To what extent these types of malnutrition may be involved in producing sterility is more or less a matter of conjecture at this time.

Through the courtesy of the Federal Emergency Drouth Relief cattle and sheep program, ample material has been at our disposal for autopsy. Approximately 100 cattle and sheep were autopsied. The parenchymatous organs of representative cases have been studied histologically. The purpose of the study was twofold: first, to ascertain by microscopic study of the tissues if an essential difference existed in the kind and severity of lesions developed in the two types of injury; second, to attempt to collect enough data to enable one, by the examination of an affected animal, to foretell the pathology already developed. The owner of an affected animal is primarily interested in knowing if his animal has fair chances of recovery, and if it has, whether the subject may become thrifty again. It has been our experience that after a severe attack of either form of poisoning an animal does but poorly. Younger animals remain stunted and appear dull. The liver is the first organ to become permanently injured. The so-called recovered cases that fail to do well subsequently also have severely injured hearts.

Gross Pathology

A. Blind staggers: A condition of atony involves the smooth muscles of the gastro-intestinal tract, gall-bladder and bladder. This condition apparently prevails in the smooth muscles of the blood-vessel walls. Congestion of the blood-vessels is observed throughout the viscera. In the winter cases of blind staggers impaction of the rumen is almost a constant finding. A stasis of food material is evident also in the omasum. The base of the papillae of the longitudinal folds or leaves of the omasum shows inflammation and even actual hemorrhage. It is not common, however, to find a degeneration of the epithelium of the omasum or of the rumen and reticulum. A dilated rumen is occasionally observed. The abomasum and the upper small intestine show varying degrees of irritation and congestion of the blood-vessels. In severe cases many petechial hemorrhages are noted, and finally there is ulceration. The large intestine, as a rule, is relatively free from irritation. The blood-vessels of the peritoneum, at

least the portion investing the intestines and abdominal organs, become congested. There is a tendency to ascites. However, the distention of the abdomen, when observed, is never a result of an abnormal amount of fluid, but is due rather to the distended stomachs. Lymph-glands are red and swollen and finally degenerate to a gelatinous, edematous mass.

The liver is acutely congested, and very early in the intoxication many areas of focal necrosis appear. Blood-cells infiltrate the area around the necrotic areas. Such necrotic areas appear as connective tissue scars, and in the contraction form pits on the surface of the organ. Cirrhosis of the liver, however, is not so prevalent as it is in alkali disease, described later. Since these congested livers are never much larger than normal, there must be active destruction of their substance from the beginning of the poisoning. The gall-bladder may be enlarged to twice its normal size. Its wall often exhibits mucoid degeneration with congestion of the blood-vessels.

There is congestion in the medulla of the kidney. Kidney stones are not uncommon in the more advanced cases. When stones occur, there is a damming back of the urine, causing occasionally large cysts. When an atrophied kidney is found, there is a compensatory hypertrophy of the other.

Petechial hemorrhages on the epicardium of the heart are common. There is also a tendency to a deposit of much fat extending down almost to the apex. There is an inflammation of the endocardium leading to hemorrhage. This inflammatory process invades the myocardium and is much more extensive around the blood-vessels. The pericardial sac often contains an abnormal amount of fluid.

The lungs are moderately congested. A variable picture is observed in the spleen. This organ is often atrophied and firm, although congestion is also noted. The ends of the long bones terminating in the hock-joints show erosion in approximately two-thirds of the cases examined. A typical case of blind staggers does not have the long, crooked hoofs.

B. Alkali disease: Compared to the pathology described above, it is evident that the lesions of alkali disease represent deteriorations which are more chronic in nature. The first abnormality observed is a dullness and a lack of vitality in the animal. At autopsy the severest injury is noted in the heart and liver. In the advanced cases the heart is invariably atrophied (so-called dish-rag heart). There are petechial hemorrhages on

the epicardium, although these are not so common as in blind staggers.

An inflammatory process extending from the endocardium into the myocardium is as prevalent as in blind staggers. The significant difference in the heart lesions in the two forms of injury is the occurrence of atrophied hearts in alkali disease. These cases do give every indication of badly decompensated hearts.

The liver, often atrophied, is cirrhotic. The surface of the organ in the very advanced cases appears scarred. The gall-bladder rarely is enlarged, although degeneration of its epithelium is as common as in blind staggers. The kidney is more seriously involved than in blind staggers, the medulla in advanced cases being hemorrhagic. Kidney stones in the calyx are rather frequent, causing a damming back of the urine and cyst formation. The spleen is atrophic and dull yellowish brown. The lungs, as in blind staggers, are congested.

The strain of too rigorous trailing on the range of the more advanced cases often causes fatal results. Nearly all typical cases of alkali disease have abnormal hoofs. The elongated hoofs may assume odd, abnormal shapes, such as one digit growing over and entirely across the other digit, or may turn upwards and grow to twice the normal length. Occasionally such digits will develop either horizontal or vertical cracks, which may reach the sensitive part of the foot. In that condition an animal exhibits pain and is reluctant to move about for grazing. The erosion of the ends of the long bones is constantly noted. In the severe, advanced cases the bone-marrow appears gelatinous.

Microscopic Pathology*

A. Blind staggers: The toxic agents which produce blind staggers cause an inflammatory reaction around the blood-vessels. A sero-fibrinous exudate containing blood cellular elements is observed.

The first injury noted in the liver cell is a cloudy swelling. In this process of congestion the nutrition of the cell is apparently deficient, as the cell shrinks, producing rather wide sinuses, especially marked around the central veins. In localized areas these widened sinuses fill with blood-cells, which must have escaped by a process of diapedesis. The next step is a necrosis of the cells in such a hemorrhage area (fig. 4). There is an evident attempt at regeneration, as bile-duct sprouts appear.

*All tissues for microscopic study were obtained from cattle.

The process of destruction (areas of necrosis) is constantly observed in all cases in which the animals had suffered severe, acute injury. So-called recovered cases that do not become thrifty again invariably show, upon the examination of the liver, connective tissue proliferation around the hepatic trinity.

A sero-fibrinous exudate is observed around the blood-vessels of the heart (figs. 1 and 2). Erythrocytes escape from the blood-vessels into the surrounding tissue. This may account for the extensive hemorrhage in the endocardium and the petechial hemorrhages on the epicardium. In the more advanced cases there are small localized areas of heavy infiltration of lymphocytes throughout the myocardium. Occasionally heart muscle atrophy is observed, although it is never so severe as in alkali disease.

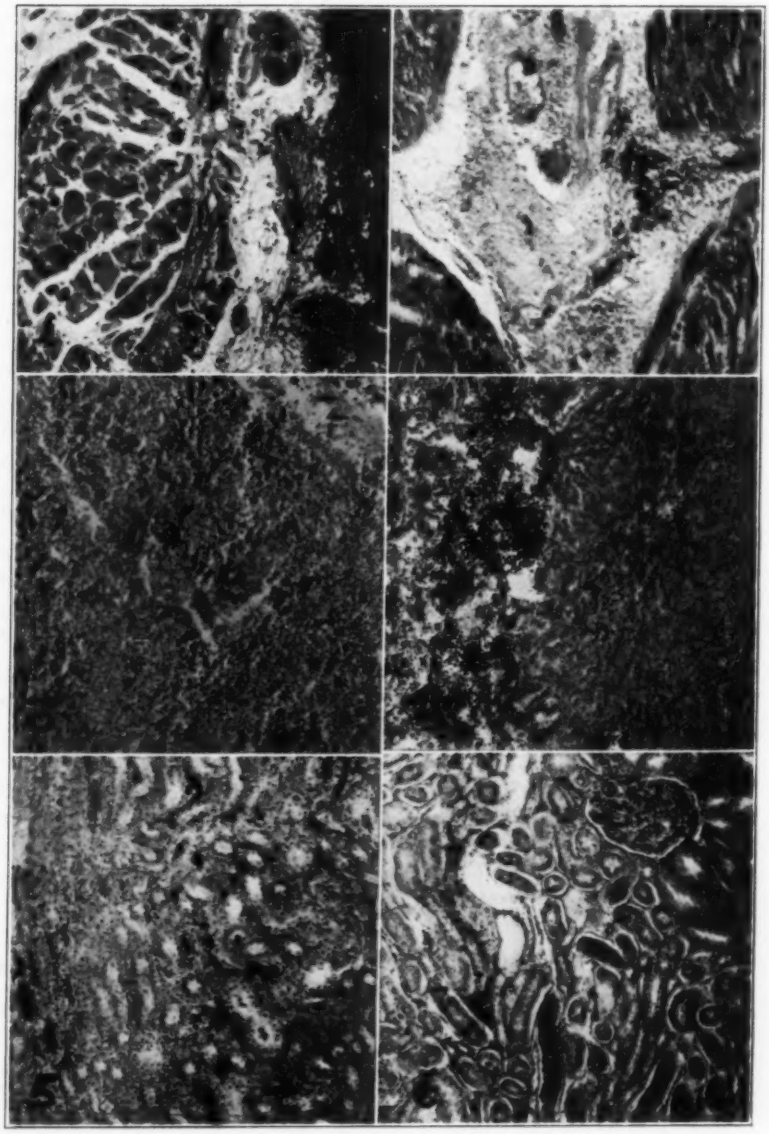
The most severe injury in the kidney is noted in the collecting tubules of the medulla. The cells at first undergo cloudy swelling. With further degeneration the epithelial cells become low, almost flat, and stain poorly. There is rather severe intertubular hemorrhage (fig. 5). In the cortex a mild glomerulonephritis is observed. The convoluted tubules may show hyaline degeneration (fig. 6), although it is not so common as in alkali disease. The spleen (fig. 3) is often congested and has a heavy deposition of hemosiderin in the more advanced cases.

In the gastro-intestinal tract not much pathology is observed before the omasum. In rare cases the rumen and reticulum may show slight inflammation. The base of the papillae of the longitudinal folds or leaves of the omasum becomes inflamed rather early in the intoxication. In severe cases this may lead to severe degeneration and necrosis of the epithelium. The abomasum is irritated, and finally petechial hemorrhages become common. Areas of desquamation of the epithelium are noted in this stomach and the upper portion of the small intestine. Farther down the intestinal tract the injury becomes less severe, and it is not common to find inflammation in the large intestine.

The lungs are congested. There is great thickening of the alveolar wall. In the cases in which the adrenals were examined

PLATE I

FIG. 1. Illustrates the inflammation in the endocardium of the heart.
FIG. 2. A typical inflammatory reaction around the blood-vessels of the heart (in the myocardium).
FIG. 3. Deposition of hemosiderin in the splenic pulp is illustrated by the dark areas.
FIG. 4. The edge of an area of focal necrosis in the liver.
FIG. 5. A portion of the medulla of the kidney illustrating hemorrhage in the collecting tubules.
FIG. 6. Illustrates hyaline convoluted tubules in the cortex of the kidney.



histologically, little pathology was observed. There is a tendency for the medulla of this gland to become hemorrhagic.

B. Alkali disease: Histologically there is little essential difference in the lesions produced by this type of injury and those of blind staggers, except that alkali disease represents a more chronic type of intoxication. Consequently *acute* injury such as *severe* irritation in the gastro-intestinal tract and other organs is not observed. Connective tissue scarring in the liver and spleen is more common.

The liver is cirrhotic and atrophied. In sheep acute yellow atrophy is not uncommon, although areas of focal necrosis are only occasionally observed. Nevertheless, in the advanced cases it is common to find the surface of the organ pitted and finely scarred with connective tissue. The liver cell is shrunken, producing wide sinuses, which are often filled with blood-cells. The greater deterioration of the cell occurs around the central veins. Around the hepatic trinity sprouting bile-ducts are observed (figs. 9 and 10). Connective tissue proliferation is common around the hepatic trinity. Infiltration of leukocytes is observed around the larger blood-vessels.

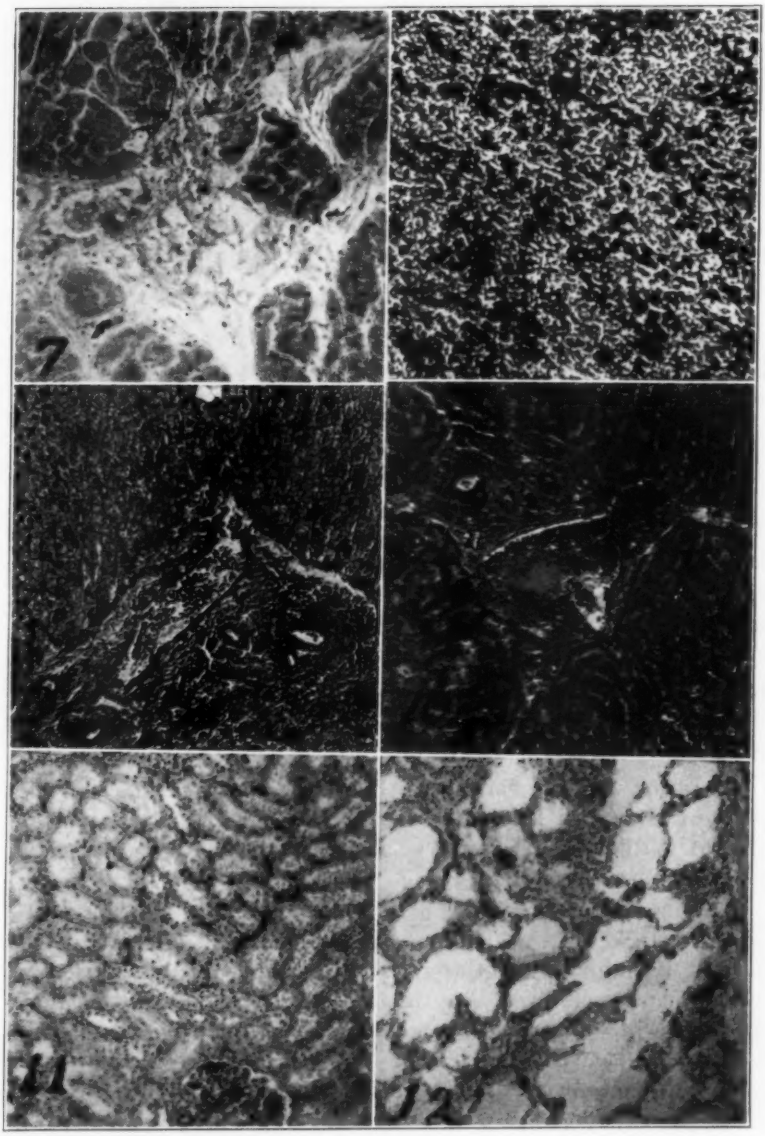
The heart is atrophied, and bundles of heart muscle fibers appear separated by an exudate of a sero-fibrinous character (fig. 7). The inflammatory process is more severe around the larger coronary vessels. Small localized areas of infiltration of lymphocytes are noted in some cases throughout the myocardium. As a rule the endocardium shows varying degrees of inflammation.

The kidney in alkali disease suffers rather severely. The injury is of the same nature as in blind staggers. The epithelial cells of the collecting tubules are low and stain poorly. There is considerable intertubular hemorrhage (fig. 11). As in blind staggers, the convoluted tubules in the cortex become hyaline, and a glomerulo-nephritis is observed.

The spleen is affected in the same manner as in blind staggers; however, the deposition of hemosiderin in the splenic pulp is

PLATE II

FIG. 7. Illustrates inflammation around a blood-vessel of the myocardium of the heart in alkali disease.
FIG. 8. The deposition of hemosiderin in the splenic pulp is very heavy, as illustrated by the dark areas.
FIG. 9. Illustrates hemorrhages and sprouting bile-ducts in the liver.
FIG. 10. Same as figure 9.
FIG. 11. Illustrates hemorrhage and severe degeneration of the tubules of the vortex of the kidney.
FIG. 12. Congestion of the lung. There is marked thickening of the alveolar wall.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heavier (fig. 8). The lesions in the gastro-intestinal tract also are of the same general character, but the intense irritation is not observed. Desquamation of the epithelium of the abomasum is observed occasionally. The lung (fig. 12) is congested as in blind staggers. The adrenals examined were hemorrhagic in both the cortex and medulla.

The sections of which photomicrographs are reproduced were stained with hemotoxylin and eosin. A Wratten H filter was used for all the photomicrographs shown. All magnifications are x 80. Figures 1 to 6 illustrate lesions produced in blind staggers, and figures 7 to 12 are those produced in alkali disease.

CONCLUSIONS

1. Data from 100 autopsies on cattle and sheep suffering from blind staggers and alkali disease indicate that the toxic principles of both types of injury have very similar physiological actions.

2. The toxicants are acutely toxic to the liver cell.

3. Blind staggers represents a more acute type of poisoning.

4. Atony of smooth muscle is rather severe in blind staggers. Regardless of the irritation of the gastro-intestinal tract, there is a stasis of food material, especially in the winter cases.

5. Kidney injury is more severe in alkali disease. Sheep suffer more from this injury than do cattle.

6. Enlarged gall-bladders are common in blind staggers and only occasionally observed in alkali disease.

7. The heart is invariably atrophied in severe cases of alkali disease.

8. Injury to the gastro-intestinal tract in blind staggers consists of irritation leading to hemorrhage and finally desquamation of epithelium. The injury is of the same character in alkali disease but milder.

9. The hearts and livers of animals suffering from an acute, severe attack of blind staggers rapidly become permanently injured. An animal so injured exhibits, upon apparent recovery, a roughened, off-color coat and is unthrifty. Many younger animals will appear runty, and due to distended stomachs appear to be bloated chronically.

10. Animals given the blind staggers treatment show immediate partial clearing of the eye.

11. Abnormal hoof growths are characteristic of alkali disease only. The erosion of the ends of the long bones is characteristic of both, but is more prevalent in alkali disease.

12. The color and the condition of the coat in poisoned cattle are in a measure diagnostic aids in recognizing this type of injury. Affected sheep have a characteristic stance and appearance; however, this type of intoxication of sheep is not so easily recognized from the behavior and the general outward appearance of the animal.

13. Although it is believed that blind staggers and alkali disease are produced by the same causative agents, nevertheless there is enough difference in the appearance and symptoms exhibited by affected live stock to retain the two terms. On the other hand, the microscopic pathology hardly justifies the designation of blind staggers and alkali disease as representing two different types of intoxication.

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To Make a Horse Like Music

An ingenious method which has been worked out by a stable sergeant in the Thirtieth Infantry to develop a liking for band music in young remounts is described by Major E. L. Nye, V. C., U. S. A., in the *Veterinary Bulletin.*

The sergeant has a radio in his room at the stables, which is noted for its loud and raucous tone. He sets the radio in the open window, ties the unsuspecting horse firmly to the outside wall of the stable, and then turns on a blast of the more audible selections. The astonished remount stands in shocked amazement at first. Then, he will faint or throw a fit, according to his temperament. His very natural objections continue with more or less violence for several days. Finally, he will quiet down and stand with dreamy eyes and pleased expression, even in the awful presence of a crooner. After two weeks, the most skeptical of remounts becomes convinced of the joys of music and appears to be unhappy without it. He is now musically educated for parade or review and will take his place with the simple dignity of the most ancient band horse.

Objection to this system of making a horse listen to a radio naturally may be raised on the ground of cruelty to animals, according to Major Nye. However, it is a situation in which something must be done, and it is believed that the results obtained justify the means, harsh as they may seem.

EQUINE ENCEPHALOMYELITIS STUDIES

1. Cross-Immunity Tests Between Eastern and Western Types of Virus*

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Indications of the existence of at least two distinct types of encephalomyelitis virus in America were pointed out by Ten-Broeck and Merrill[1] and by the writers,[2] in 1933. Records and Vawter,[3] in 1934, corroborated these findings in the case of infection in guinea pigs but concluded that the results of cross-exposure tests in horses did not support the earlier evidence of plurality of virus types. Their results were presented as suggestive of actual cross-immunity between eastern and western virus infection; the fact of the greater virulence of eastern strains was recalled and offered as the essential difference in the strains of virus which had been recovered in the geographically separated outbreaks.

In the course of the Bureau's studies of encephalomyelitis, several groups of horses immune to one or the other type of virus have become available for cross-immunity tests. These animals before immunization were all discharged U. S. Army horses, of various weights and ages, which, except for lameness or other disabling (for service) abnormalities, were so selected as to be calculated to be free of any disease which would be likely to disturb the accuracy of experimental results. As far as was known, they had not suffered an attack of encephalomyelitis nor had they been exposed by contact with either affected animals or premises on which the disease was known to exist.

In table I a group of animals, including a calf and a sheep which had been reared on the Experiment Station farm at Bethesda, Md., all of which had been immunized against or had resisted experimental exposure to western virus, are shown to have been exposed to eastern virus by intracerebral inoculation.

The western virus consisted of a strain recovered by the authors from a South Dakota case in 1932, and the eastern virus was from a Maryland case, also recovered by the authors, in 1933. Both of them were used in tests I, II, III and IV.

It is believed that these results clearly indicate, in the animals tested, that immunity to eastern virus did not follow or result

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TABLE I—*Cross-immunity test: Resistance of western immunes to eastern virus.*

ANIMAL	HISTORY OF EXPERIMENTAL EXPOSURE TO WESTERN VIRUS			INTRACEREBRAL INOCULATION OF EASTERN VIRUS	
	METHOD	DATE	RESULTS	DATE	RESULTS
Horse 743	Intracerebral Intranasal	8–18–33 10–11–33	Survived No symptoms	2–9–34	Encephalomyelitis; killed 5th day
Horse 735	Mosquitoes Intradermic Intranasal Intracerebral	8–7–33; 8–8–33 9–22–33 10–11–33 11–23–33	Survived No symptoms No symptoms No symptoms	2–27–34	Encephalomyelitis; killed 5th day
Horse 747	Subcutaneous injections of formalin-killed guinea-pig-brain virus Intracerebral	12–9–33; 12–20–33; 4–10–34; 4–20–34; 4–30–34; 5–14–34 6–11–34	 Survived	9–7–34	Encephalomyelitis; killed 3rd day
Calf 1855	Intracerebral Intracerebral	5–9–33 11–10–33	Survived No symptoms	2–28–34	Encephalomyelitis; survived*
Sheep 1073	Intracerebral Intracerebral	5–9–33 11–10–33	No symptoms No symptoms	2–28–34	Encephalomyelitis; dead 6th day

*A subsequent exposure to eastern virus produced no noted evidence of disturbance in this animal.

Note: The remaining unused portions of the western virus suspensions used in these inoculations produced typical encephalomyelitis when injected intracerebrally in control animals of a susceptible species.

from the experience of, or resistance to, western virus infection. Before accurate conclusions could be drawn, it was necessary to use the opposite order of exposure to the two types of virus. Admitting the greater virulence of the eastern virus, as indicated by a more acute course and greater mortality than is usually produced by western type virus, it was still possible that there was no actual immunological difference between the strains of different origin. The availability of a considerable number of eastern immunes, from time to time, permitted such tests and the results of cross-exposure to western virus, by intracerebral injection, are presented in tables I, II, III and IV.

TABLE II—*Cross-immunity test: Resistance of eastern immunes to western virus.*

HORSE	HISTORY OF EXPERIMENTAL EXPOSURE TO EASTERN VIRUS		INTRACEREBRAL INOCULATION OF WESTERN VIRUS	
	METHOD	DATE	DATE	RESULTS
762	Intradermic	2–27–34	10–3–34	Encephalomyelitis; chronic ataxia
774	Subcutaneous Intracerebral	7–27–34 9–7–34	10–3–34	Encephalomyelitis; slow, incomplete recovery
775	Subcutaneous Intracerebral	7–27–34 9–7–34	10–3–34	Encephalomyelitis; recovery
754	Intranasal Intracerebral	2–27–34 9–7–34	10–3–34	Encephalomyelitis; slow, incomplete recovery
779	Formalin-inactivated horse-brain virus Subcutaneous Intracerebral	6–15–34; 6–25–34 7–27–34 9–7–34	10–3–34	Encephalomyelitis; recovery
783	Subcutaneous Intracerebral	7–27–34 9–7–34	10–3–34	Encephalomyelitis; recovery
794*	Control		10–10–34	Encephalomyelitis; killed 6th day

*The same saline suspension of virus which had been used 10-3-34 and which until 10-10-34 had been stored in the refrigerator was used in the exposure of this animal. The animal was killed when down in a hopeless condition on the 6th day following inoculation.

Note: In all instances, the eastern virus which the above animals received was simultaneously checked by intracerebral or other method of injection of one or more horses and by intracerebral inoculation of guinea pigs.

Inasmuch as the foregoing test (table II) was open to the criticism that the control animal had not been exposed simul-

taneously with the immune animals, even though the identical virus sample was used later, and since it was accepted that the results might indicate that the eastern immune animals had acquired a resistance, if not complete immunity, against western type virus, additional tests were seemingly advisable. It had previously been observed that horses which survived the injection of active virus by any of several methods, other than intra-

TABLE III—*Test of immunity to heterologous and homologous virus of animals exposed to eastern virus by tongue inoculation.*

Horse	Experience with Eastern Virus			Virus Used in Intracerebral Exposure 11-16-34	Results
	Source	Method	Date		
803	Brain*	Intramuscular	9-7-34	Western	Encephalomyelitis; killed 3rd day
787	Pad†		10-13-34		Remained normal
819	Formalin-inactivated horse brain‡		9-10-34; 9-20-34		
	Pad		10-31-34		Encephalomyelitis; killed 6th day
826	Pad		10-31-34		Encephalomyelitis; permanent disability
804	Pad		10-31-34		Encephalomyelitis; killed 3rd day
827	Control				Encephalomyelitis; killed 3rd day
814	Brain	Intramuscular	9-7-34	Eastern	Remained normal
784	Pad		10-13-34		Remained normal
810	Formalin-inactivated horse-brain‡		9-10-34; 9-20-34		Remained normal
	Pad		10-31-34		
817	Pad		10-31-34		Remained normal
816	Formalin-inactivated horse-brain		9-10-34; 9-20-34		
	Pad		10-31-34		Remained normal
801	Pad		10-31-34		Remained normal
812	Control				Encephalomyelitis; killed 3rd day

*Virus in the form of guinea-pig brain intramuscularly in the tongue.
†Virus in the form of guinea-pig pads, showing lesions following intradermic inoculation of plantar skin, intramuscularly in the tongue.
‡Subcutaneous injection; all others intramuscular.

cerebral, were quite uniformly immune against an intracerebral exposure to homologous virus. Among these methods of virus introduction was the intramuscular injection in the tongue, and a number of animals so treated were available. Since these particular animals had not been subjected to an intracerebral test of immunity, it was decided to expose the essentially identical animals alternately to homologous and heterologous strains of virus. All the animals had, prior to the proposed test, been treated with a virus of proved activity, which in three instances had been preceded by treatment with formalin-inactivated horse brain suspensions. This test is recorded in table III.

In the conclusion of this study, the animals which had shown no symptoms following the intracerebral inoculation of eastern virus, as shown in table III, and an additional animal which had survived an intramuscular injection in the tongue with guinea-pig-pad virus (eastern), were exposed as shown in table IV by the intracerebral injection of western virus.

Virus Used

In all four of the cross-immunity tests, the virus used in the intracerebral injections consisted of a suspension in normal saline solution, of guinea-pig brain. This was preferably used fresh, rather than brain which had been stored in the refrigerator in glycerin solution. With the exception of sheep 1073 in table I, those animals which, following the typical encephalomyelitis syndrome, were down and unable to rise, were killed for humane and other reasons, it being practically a foregone conclusion that animals which reached such a state very rarely recover, at least when not therapeutically assisted. The brains of killed animals appeared to be more likely to be culturally sterile and in other ways more desirable sources of virus for other studies than those from animals which had died from the disease. In these tests it may be observed that animals, previously shown, by intracerebral inoculation, to be immune to the same type of virus which was used in the concluding cross-exposure in each test, are not provided. Our experience and, as far as we are aware, that of others do not indicate that an animal which has withstood a primary intracerebral inoculation of active virus of a given type will, within any reasonable period, fail to resist any subsequent, moderate exposure of like nature to the same virus.

Discussion

The above tests appear to justify the conclusion that the two strains of virus which were used in these tests are immunologically distinct, not only in the guinea pig, as had been indicated previously,[1-3] but in the horse as well.

The eastern strain of virus which was used is but one of sixteen which the writers have recovered from cases in New Jersey, Delaware, Maryland and Virginia, in 1933 and 1934. But, in guinea pigs, there has been no evidence that any one of these

TABLE IV—*Cross-immunity test: Resistance of eastern immunes to western virus.*

HORSE	EXPERIENCE WITH EASTERN VIRUS			RESULTS OF INTRACEREBRAL INOCULATION OF WESTERN VIRUS (12-24-34)
	SOURCE	METHOD	DATE	
824	Pad	Intramuscular (tongue)	11–9–34	Enceph. K. 5th day
814	Brain	Intramuscular (tongue)	9–7–34	
		Intracerebral	11–16–34*	Enceph. K. 5th day
784	Pad	Intramuscular (tongue)	10–13–34	
		Intracerebral	11–16–34*	Enceph. Progressing convalescence
810	Formalin-inactivated brain	Subcutaneous	9–10–34; 9–20–34	
	Pad	Intramuscular (tongue)	10–31–34	
		Intracerebral	11–16–34*	Enceph. K. 5th day
817	Pad	Intramuscular (tongue)	10–31–34	
		Intracerebral*	11–16–34	Enceph. K. 5th day
816	Formalin-inactivated brain	Subcutaneous	9–10–34; 9–20–34	
	Pad	Intramuscular (tongue)	10–31–34	
		Intracerebral*	11–16–34	Enceph. K. 5th day
801	Pad	Intramuscular (tongue)	10–31–34	
		Intracerebral	11–16–34*	Enceph. K. 6th day
830	Control			Enceph. K. 5th day

*See table III.

several strains was different, immunologically, from the others. Also, all 16 strains apparently have been of the same virulent nature.

The western strains, fewer in number but apparently similar each to the others in pathogenicity and immunity reactions in guinea pigs, may apparently likewise be similarly grouped in one class. These strains have included three recovered by the writers from South Dakota cases, one each in 1932, 1933 and 1934, one from California supplied by Dr. C. M. Haring and one from Utah supplied by Dr. D. E. Madsen.

Guinea pigs immunized against the Maryland virus, which has been used in the tests in horses, have consistently been immune against an attempted superimposed infection with any of the

fifteen other strains of eastern origin and have, with infrequent exceptions, been shown to be susceptible to exposure to any of the five western strains.

The representative western strain produces an immunity which prevails against other western strains but usually fails to protect against any of our eastern strains. It has been observed that occasionally guinea pigs immune to one type of virus may not succumb to an encephalomyelitis following heterologous virus exposure.

It is presented that the actual death of an animal, attended by a wholly typical encephalomyelitis, may not be absolutely essential in the determination that the animal in question is susceptible. The writers believe that a rise in temperature, attended by lassitude and such other signs of incipient infection as icterus, may be the only indication that the animal was susceptible and has actually contracted the infection. Such indications of infection have not been observed in animals specifically immune against virus of homologous type but have been encountered in animals exposed to virus of a type which has not been previously experienced by the animals in question and this may occur not only in animals immune to one type when exposed to the other of the present known types, but in normal animals as well.

The writers have record of the exposure of a normal horse (one never known to have been exposed to encephalomyelitis virus, certainly not experimentally) to the bites of western virus-infected mosquitoes, which animal, though never showing a temperature of over 101.0° F. or more than very mild indisposition, was shown to carry virus in its blood intermittently for a period of at least 72 hours. Although under observation for nearly three months since that time, no characteristic evidence of encephalomyelitis *per se* has developed. And yet it must be said that the animal was susceptible and was infected.*

The presence of virus in the blood has been found following other methods of exposure, as previously reported,[4] and has subsequently been repeatedly demonstrated by the writers with such regularity that it seems to be an essential part of the picture of encephalomyelitis infection, certainly in experimental cases and assumedly in natural infection.

The appropriateness of the terms "eastern" and "western," as applied to types of encephalomyelitis virus, may be questioned. It appears to be true, however, that the two have been geo-

*A detailed report of this case will be made at a future time, in coöperation with the Bureau of Entomology.

graphically separated up to the present time and the terms seem to have a use in the United States, until such time as other more characteristic features are determined. Study of the actual nature of the virus and the disease itself may or may not serve to differentiate the two apparent types of virus which produce, as far as has been reported, an essentially identical infection. Possible meteorological or biological influences upon virus types are as yet, of course, only conjectural but may eventually be determined as factors affecting type stability.

SUMMARY

Evidence of the existence of two immunologically different types of equine encephalomyelitis virus is reviewed and cross-immunity tests in horses tending to confirm previously reported observations in guinea pigs that eastern and western viruses are of two distinct types are presented. Three western-virus-immune horses exposed intracerebrally to eastern virus developed typical encephalomyelitis and were destroyed *in extremis*. With one exception, 18 eastern immunes inoculated intracerebrally with western virus developed typical encephalomyelitis and of these nine were sacrificed when in a hopeless condition, while eight which did not become prostrate were allowed to continue in the experiment, three making incomplete recoveries and five partial or delayed recoveries.

A calf immune to western virus manifested a typical encephalomyelitis syndrome following intracerebral exposure to eastern virus. A sheep which had resisted two intracerebral inoculations of western virus without any noticeable reaction developed typical encephalomyelitis and died.

That occult cases of encephalomyelitis may occur amongst experiment animals is illustrated by the case in which a horse, exposed to bites of western-virus-infected mosquitoes, developed only a mild indisposition without a true febrile reaction but was shown to carry virus in its blood intermittently for at least 72 hours.

The designation of the two apparent types of encephalomyelitis virus as "eastern" and "western" may be objectionable, but the use of the terms is suggested as advisable, at least in the United States, until such time as further studies may reveal more specific differential characteristics.

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Errata

In the paper, "Some Aspects of Infection and Immunity in Equine Encephalomyelitis," by M. S. Shahan and L. T. Giltner, published in the June, 1934, issue of the JOURNAL, on page 930 the statement reading, "Of six horses inoculated subcutaneously with 5 to 10 cc of 20 per cent eastern virus," should read "of 2.0 per cent eastern virus." On page 933, in table II, the dates should have been 2-26-34 and 3-8-34 instead of 2-26-33 and 3-8-33. Again, on this page, in the footnote (*) under table II, 20 per cent virus is stated to have been used, whereas 2.0 per cent virus was used.

Doctor Eichhorn on Program of London Congress

Dr. Adolph Eichhorn (N. Y.-Amer. '00), director of the Veterinary Department of the Lederle Laboratories, Pearl River, N. Y., will be one of the prominent American lecturers on the program of the Second International Congress for Microbiology, to be held in London, July 27 to August 1, 1936. Dr. Eichhorn will discuss "The Prophylaxis and Serum Treatment of Human and Animal Diseases Caused by Anaerobic Bacteria." The invitation was extended by Dr. R. St. John-Brooks, of the Lister Institute. The first Congress was held in Paris several years ago and, on that occasion, several eminent microbiologists from the United States acted as reporters.

Virginia Planning for Big Meeting

The Virginia State Veterinary Medical Association will hold its 42nd annual meeting in Richmond, July 10-12, 1935, and has invited the state associations of Maryland, North Carolina and West Virginia, and the veterinarians of the District of Columbia to take part in the celebration. The guest of honor at the joint meeting will be Dr. R. S. MacKellar, president of the A. V. M. A., who will bring a message from the national association. Dr. H. H. Rowe, of Richmond, is chairman of the Committee on Local Arrangements.

EQUINE ENCEPHALOMYELITIS CROSS-IMMUNITY IN HORSES BETWEEN WESTERN AND EASTERN STRAINS OF VIRUS*

Supplemental Report

By EDWARD RECORDS *and* LYMAN R. VAWTER
Nevada Agricultural Experiment Station, Reno, Nev.

In a recent paper,[1] we reported that six out of eight horses, immune to the western type of equine encephalomyelitis virus, were also resistant to a Delaware strain of the eastern type of virus. In this paper, one control horse (9663) was reported to be convalescing from severe symptoms following intranasal instillation of Delaware virus. This individual recovered in about six weeks without apparent disability. On July 23, 1934, sixty days after receiving the Delaware virus, this horse was injected intracranially† with 5 cc of a 2 per cent suspension of a highly virulent western virus and survived without developing symptoms of encephalomyelitis.

Three control guinea pigs, injected intracranially with the same western virus suspension, were either dead or moribund on the fifth day after injection. The m.l.d. of this particular virus suspension was 0.2 cc of a 1:1,000,000 dilution for 400-gram guinea pigs.

In August, 1934, we received a strain of equine encephalomyelitis virus obtained by Dr. D. E. Madsen, of the Utah Agricultural College, during an outbreak of this disease in Cache Valley, Utah, in July, 1934. The virus as received from Dr. Madsen represented the first guinea-pig passage. This Utah virus was passaged twice in guinea pigs immediately upon reaching us and was found to be of low virulence, as paralysis and prostration did not occur until the sixth or seventh day following intracranial injection.

Serum-protection and cross-immunity tests made with guinea pigs indicated that this Utah virus was immunologically similar to the other strains of western virus of equine encephalomyelitis previously examined.

Incident to other experimental work, which is being reported in a separate paper, four horses were exposed to infection with this Utah virus.

It is believed that all of the horses used in this experiment were susceptible to infection, as they originated from a range

*Received for publication, April 4, 1935.

†All intracranial inoculations were made under general anesthesia.

district in northern Nevada where equine encephalomyelitis has not been known to occur. The history of each horse injected with Utah virus and one contact control horse is briefly summarized as follows:

Horse 126 was exposed on August 22, 1934, by about ten punctures made into the skin on the lateral aspect of the upper lip by means of a 20-gauge needle which had been dipped in virus suspension. Mild symptoms, such as high fever, depression, lip droop and triceps tremor, appeared on the eighth day and persisted until the twelfth, followed by rapid return to normal.

Horse 125 was given a single intranasal instillation of 5 cc of a 2 per cent suspension of brain virus on the same date. Depression and high temperature appeared on the sixth day followed by muscle tremors, lip droop and slight ataxia. These symptoms persisted until the 13th day, followed by slow recovery.

Horse 136, which was injected intracranially with 5 cc of a 2 per cent suspension of Utah virus, developed the usual symptoms of encephalomyelitis and died on the seventh day after prostration lasting only a few hours.

Horse 131, an uninoculated control, showed mild symptoms such as high fever, congestion of conjunctiva, depression and slight lip droop on the tenth day of contact in the same corral with horse 125. The symptoms were typical of a mild abortive attack of encephalomyelitis and subsided entirely in three days, followed by recovery. It appears that infection resulted from close contact with horse 125.

Even though the recently captured Utah strain of virus manifested a low degree of virulence for horses and guinea pigs upon intracranial or intranasal inoculation, it readily caused infection in horse 126 by cutaneous pricking with a 20-gauge needle which had been dipped in virus. The dosage in this instance was only a very small fraction of that used for intranasal and intracranial inoculation of other horses.

Blood was collected from horses 125 and 136 at the sixth hour after exposure to virus and daily thereafter until the subsidence of symptoms in horse 125 and the death of horse 136. The blood specimens were allowed to clot and then were centrifuged lightly. The serum was injected into two guinea pigs, each receiving 0.3 cc intracranially and 1 cc subcutaneously, on the day of collection.

Virus first appeared in the blood of the intranasally infected horse 125 at the 48th hour and persisted until the 144th hour, in amounts infective for guinea pigs. Horse 136, which was in-

jected intracranially, carried virus in the blood from the 48th to the 120th hour. This same strain of Utah virus, injected intracranially into guinea pigs, did not appear in the blood until the 48th hour after injection and was still present until the 96th hour, but absent thereafter.

We have always suspected that the mucoid nasal discharge common to this disease in horses might carry virus. However, repeated attempts to demonstrate virus in the Berkefeld filtrates and nasal washings have been negative.

As a further check on this angle, nasal washings were collected also from horses 125 and 136, six hours after virus exposure and daily thereafter. After the head was secured in a rigid position favorable for free drainage from the nostrils, the muzzle and external nostrils were carefully cleaned with moist cotton swabs.

Forty to 50 cc of buffered salt solution (pH 7.4) was introduced along the median septum on each side by means of a soft rubber catheter attached to a 40-cc syringe and the return flow caught in a sterile pan. The recovered nasal washings amounting to 30 to 50 cc were turbid and mucoid but free of blood. After being strained through sterile gauze and centrifuged for one hour at high speed, the clear supernatant fluid was injected into guinea pigs in the same manner and amounts as the blood serum, Berkefeld filtration being omitted.

The nasal washings of horse 125, intranasally infected, contained virus at the 72nd and 96th hours only. We were surprised that virus was not detected in the washings taken at the 6th and 24th hours after the intranasal instillation of virus. Horse 136, injected intracranially, carried virus in the nasal washings at the 72nd and 96th hours only.

The criteria for judging the presence of virus in either blood-serum or nasal washings were the usual virus symptoms in the guinea pigs; namely, high fever, loss of weight, and paralysis. Several guinea pigs showed typical virus infection temperature curves, but survived. These were subsequently found to be solidly immune to the intracranial injection of virus.

The prolonged presence of virus in the blood of the two horses experimentally infected with the recently captured Utah virus and the concurrent discharge of virus in the nasal secretions, could afford unusual opportunity for transmission by insect vectors and possibly by contamination of equipment.

In September, 1934, we received through the courtesy of Dr. J. H. McNeil, of Trenton, New Jersey, half a horse brain from

a natural case collected during a current outbreak of equine encephalomyelitis in that state. This New Jersey virus proved extremely virulent for guinea pigs upon intracranial injection, causing prostration and death in about 65 hours.

On October 11, 1934, approximately 45 to 50 days subsequent to infection with the Utah virus, the three surviving horses were each injected intracranially with 4 cc of a 2 per cent suspension of the New Jersey virus, representing the second guinea-pig passage. Horses 125 and 126 survived without symptoms; horse 131 was prostrate and moribund at the 75th hour after injection and was destroyed.

Control guinea pigs injected intracranially with this same suspension of New Jersey virus were all dead at the 65th hour. The New Jersey virus behaved similarly to the Delaware virus in that the course of the experimental disease in both horses and guinea pigs was about 48 hours less than is ordinarily the case with western strains of virus under similar experimental conditions.

Blood samples were obtained from horses 125 and 126 for virus neutralization test on the 28th day after exposure to New Jersey virus. The details of this test were essentially the same as reported in another paper.[2]

The serum of both horses completely neutralized the Nevada virus. Some protection was shown against New Jersey virus as the serum of horse 125 fully protected one guinea pig and delayed infection in another, so that death did not occur until the seventh day.

The serum of horse 126 retarded infection with New Jersey virus so that death was delayed until the seventh and eighth days.

The two control guinea pigs which received New Jersey virus were both dead in 90 hours. The Nevada virus controls died on the fifth and sixth days after inoculation.

DISCUSSION

To date we have conducted cross-immunity tests on eleven horses which were presumably immune to western virus either as a result of deliberate immunization or survivors to the experimentally induced disease. Eight of these eleven horses were also resistant to the eastern type (Delaware and New Jersey) of encephalomyelitis virus.

The failure of horse 131 to survive the immunity test with New Jersey virus raises the question as to whether or not the symptoms shown were positively due to virus infection. How-

ever, two other horses which were known to be resistant to western virus failed to survive when subsequently exposed to eastern virus.

Horse 9663, which recovered from a severe attack of encephalomyelitis induced by intranasal instillation of Delaware virus, was subsequently resistant to intracranial injection of virulent western virus.

Serum-protection and cross-immunity tests conducted with guinea pigs alone would imply that the eastern and western strains of equine encephalomyelitis virus could be placed in separate immunologic groups. Our results in connection with guinea pigs, using virus originating in the United States, are similar to those reported by TenBroeck and Merrill,[3] Giltner and Shahan,[4] and Howitt.[5]

It now appears, from cross-immunity tests on horses, using both established stock strains and recently recovered strains with limited foreign-host passage, that the differences between the western and eastern types of virus may be only different degrees of virulence rather than specific immunologic dissimilarity.

The virus of equine encephalomyelitis apparently not only may enter the equine host through the nasal mucosa but may also be discharged from this region while the virus is present in the blood.

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Doctor Inoue Visits Veterinary Centers

Dr. T. Inoue, of the Mukden Institute for Infectious Diseases of Animals, South Manchuria Railway Company, Mukden, Manchoukuo, is visiting centers of veterinary activity in the United States. He called at the A. V. M. A. office in Chicago, about the middle of May, on his way East. On his journey from the Pacific Coast, Dr. Inoue visited the University of California, Colorado Agricultural College and Kansas State College. Following his stay in Chicago, he planned to visit points of interest in Detroit, Washington and New York.

RESULTS OF INOCULATING LABORATORY ANIMALS WITH EQUINE BRAIN-TISSUE SUSPENSIONS AND EQUINE BRAIN-TISSUE FILTRATES FROM SPONTANEOUS CASES OF SO-CALLED CORNSTALK DISEASE*

By ROBERT GRAHAM, *Urbana, Ill.*
Division of Animal Pathology and Hygiene

During the late fall and winter of 1934-35, an acute and fatal encephalomyelitis of horses appeared in central Illinois. It is estimated from available reports that more than 5,000 horses succumbed to this or clinically indistinguishable maladies. On a few farms all the horses succumbed. More often, however, only one to four cases developed, the remaining horses on the premises continuing to remain apparently healthy. On recognition of the disease, the rations were changed when other feed was available. Prompt eliminative and stimulative treatment of affected animals in the early stages was frequently followed by recovery, although well-developed cases of the disease succumbed notwithstanding vigorous, symptomatic treatment.

The symptoms in affected horses appeared to point to an involvement of the encephalon and cord, while epizoölogically the causative factor or factors, as in two previous enzoötic outbreaks occurring in Illinois in 1893 and 1914, were apparently associated with the feeding of damaged corn or with the pasturing of horses on cornstalks. In the previous outbreaks as well as that of 1934-35, a dry summer season preceded a rainy wet fall. Since a majority of the horses that developed the disease in Illinois in 1934-35 were pastured in cornfields or fed 1934 damaged corn, the encephalitic syndrome was commonly referred to by clinicians and stockmen as "cornstalk disease." The malady was known also as forage poisoning, blind staggers, encephalomyelitis, cerebrospinal meningitis, and cerebritis.

Studies at the Illinois Agricultural Experiment Station on the 1934-35 outbreak in central Illinois afforded an opportunity to examine the encephalons of more than 50 horses that had succumbed or were destroyed in a moribund condition. Of the brains examined, 22 originated in Champaign County; six in Douglas; four each in Iroquois and DeWitt; three each in Ford, Piatt and Macon; two each in Will, Montgomery and Christian; and one each in Macon, Sangamon and Woodford counties.

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The possibility of the presence of a filterable virus involving the encephalon prompted the inoculation into laboratory animals of horse brain-tissue suspensions and brain-tissue filtrates (Berkefeld N and Mandler) of naturally affected horses. These inoculations were made intracerebrally into guinea pigs, pigeons and mice, and subcutaneously into guinea pigs, pigeons and rabbits. Guinea pigs, pigeons and rabbits also were injected either subcutaneously or intracerebrally with unfiltered and filtered brain tissues of laboratory animals that succumbed following the injection of horse brain-tissue suspensions or horse brain-tissue filtrates. Horse brain-tissue suspensions that proved to be highly contaminated with bacteria, as judged by growth from seeding on plain agar and incubating 24 to 48 hours at 37° C., were injected into the foot pads of guinea pigs, while guinea pig brain-tissue suspensions as well as suspensions of brain-tissue from pigeons that succumbed were composited in nutrient broth

TABLE I—*Equine brain-tissue suspension and filtrate inoculations. (Partial summary.) All results negative.*

Brains	Suspensions or Filtrates*	Animals Inoculated		Method of Inoculation
		Kind	Number	
20	S22	Rabbits	22	
33	S34	Guinea pigs	46	
34	S30	Pigeons	30	Subcutaneous
11†	S 9†	Rabbits, guinea pigs or pigeons	17	
15	S10	Guinea pigs	62	
8	S 4	Pigeons	8	
8	S 4	Mice	16	
2	S 1	Horses	2	Intracerebral
2†	S 1†	Horses	2	
16†	S 8†	Guinea pigs	29	
1	S 1†	Mice	4	
13	S 5	Guinea pigs	18	Footpad
13†	S 4†	Guinea pigs	16	
20	F12	Rabbits	12	
20	F12	Guinea pigs	12	
20	F12	Pigeons	12	Subcutaneous
6†	F 3†	Rabbits, guinea pigs or pigeons	9	
31	F23	Guinea pigs	65	
2	F 1	Horse	1	Intracerebral
7†	F 3†	Guinea pigs	7	

*S=Suspension. F=Filtrate.
†From rabbits, guinea pigs or pigeons which had been inoculated with horse-brain suspensions.

and injected intracerebrally into two horses. Table I shows a partial summary of inoculation experiments.

Many guinea pigs injected with unfiltered equine brain-tissue suspensions died following intracerebral or subcutaneous inoculation. The period of incubation, the symptoms in fatally inoculated guinea pigs, and the negative thermal reactions failed to suggest the presence of the western equine encephalomyelitis virus as described by Meyer, Haring and Howitt,[1] and Records and Vawter,[2] or of the eastern virus as described by Giltner and Shahan.[3] The inoculation with brain-tissue filtrates was followed by a lower mortality than the inoculation with unfiltered tissue suspensions. The symptoms preceding death in the guinea pigs were not suggestive of equine encephalomyelitis, eastern or western type. The technic employed in inoculating horse brain-tissue suspensions and filtrates from cases of so-called cornstalk disease was checked by simultaneously inoculating intracerebrally into guinea pigs known eastern and western equine encephalomyelitis brain-tissue viruses obtained from the Pathological Division of the U. S. Bureau of Animal Industry. The results in the latter tests were positive. In this connection the specificity of eastern and western equine encephalomyelitis antisera was demonstrated in guinea pig protection tests as noted by TenBroeck and Merrill.[4]

The results of inoculating laboratory animals with equine brain-tissue suspensions and equine brain-tissue filtrates from horses that died of so-called cornstalk disease lends evidence to support the contention that the type of equine encephalomyelitis which occurred in Illinois in 1934-35 probably is not associated with either the eastern or the western virus of equine encephalomyelitis.

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Be Colorful

To avoid a colorless existence, keep in the pink of condition, do things up brown, treat people white, be well read, and get out on the green under the blue frequently.—*Medical Times.*

ASPERGILLOSIS OF WILD TURKEYS REARED IN CAPTIVITY*

By A. J. DURANT† *and* C. M. TUCKER‡
University of Missouri, Columbia, Mo.

Since Mayer and Emmet, in 1815, recorded the presence of Aspergillus in the lungs of a jay, the fungus has been reported frequently from all the avian families in Europe and the United States. Domestic fowls and wild birds in captivity are often affected by aspergillosis caused, usually, by *A. fumigatus* Fres. The disease is characterized by the development of the fungus in the respiratory organs.

The Missouri Fish and Game Commission began propagating wild turkeys in a state game preserve in 1934. During the spring, there occurred a severe loss of poults which proved attributable to aspergillosis originating from a commercial mash feed infected by *A. fumigatus*. Since this is probably the first record of a serious epizoötic among wild turkeys under conditions of artificial propagation, it seems worth while to report the outbreak in some detail. Aspergillosis is not common among poultry in Missouri and the senior author has observed only three cases during 15 years.

In May, 1934, the propagation of wild turkeys was started in a location and in new buildings where turkeys had not been grown. The first group of 385 day-old poults in a new brooder-house was fed the commercial mash. The disease appeared at five days of age and the mortality rate reached its maximum at about 15 days. At three weeks, the epizoötic had subsided. A second group of 400 poults suffered more severely than the first, the high mortality rate extending to more than three weeks after hatching. Of the 785 poults, fewer than 200 survived, and most of the survivors were from the first group hatched (fig. 1).

In reporting the disease, the superintendent stated that the birds became sick and died in convulsions within 24 hours. Pul-

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†Veterinary Department.
‡Botany Department.

lorum disease was at first suspected, since the symptoms among the dying five-day poults included the presence of a whitish discharge from the vent. There was some coccidiosis infection in both groups, a factor which probably complicated the normal course of the disease and affected recovery.

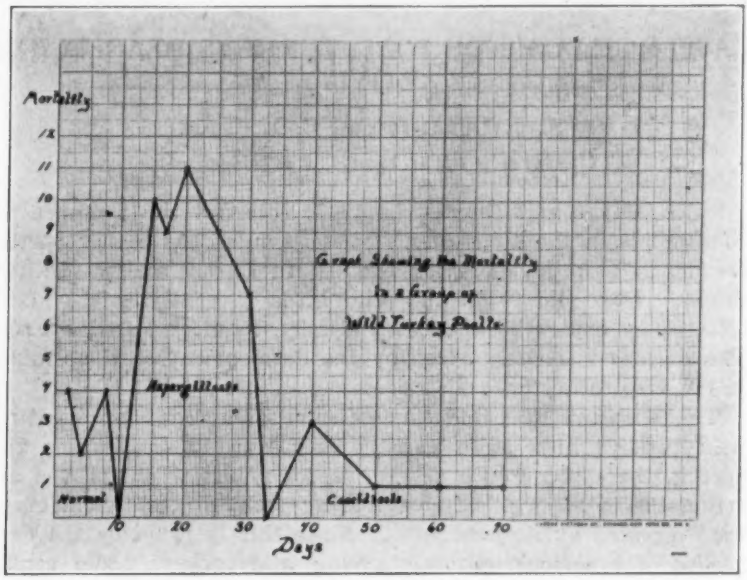

FIG. 1. Graph of the mortality rate in a group of young wild turkeys. The mortality is attributable largely to aspergillosis.

Postmortem examinations of birds of three age groups revealed infection of the lungs and adjacent air-sacs in all. The lesions in the lungs of the older (three to four weeks) birds were apparently chronic, small, yellowish-white nodules of tubercles, closely resembling the residue of the subcutaneous mite found under the skin in adult hens. The nodules were abundant throughout the lungs and varied from minute, barely visible points to tubercles 6 to 8 mm long and 3 to 5 mm in diameter. In the five- to ten-day-old poults, the lesions were more acute. The lungs were dark red and congested and stippled with minute tubercles.

It appears that the formation of large tubercles indicates a chronic form of infection and suggests a considerably greater degree of resistance than is manifest in the younger birds showing the red or congested lung condition. Microscopic examination of lung tissue from the latter showed complete penetration by

the septate hyphae of the fungus, while the lungs of older birds with large tubercles and no congestion revealed no hyphae in the tissues around the tubercles. The presence of hyphae could be detected only in the tubercles. No spore formation by the fungus was observed in the lungs.

Isolations from congested lung tissue on acidified potato dextrose agar yielded an aspergillus (figs. 2 and 3). Cultures sent to Dr. Charles Thom were identified as *A. fumigatus* Fres.

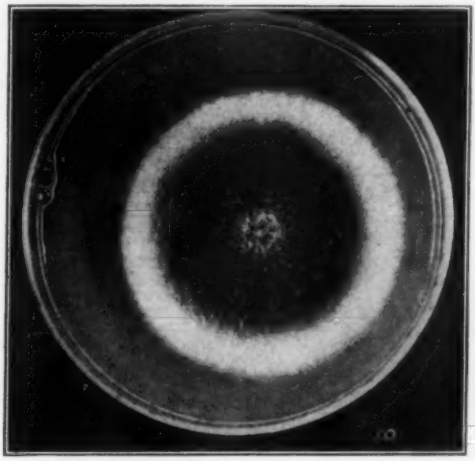

FIG. 2. A pure culture of *Aspergillus fumigatus* Fres, isolated from lung tissue of a wild turkey poult. (Potato dextrose agar.)

Samples of the commercial mash feed proved heavily infected by *A. fumigatus*, and approximately 75 per cent of the fungus mycelia developing in agar plate isolations were of this species. In the laboratory, two young poults were fed this mash. One died after three days from an undetermined cause. The second died after ten days with symptoms similar to those of the birds in the infected flocks, including the typical reddening of the lungs and the formation of minute tubercles. *A. fumigatus* was isolated from the lung tissues.

Large flasks of the infected mash were autoclaved at 20 pounds pressure for three hours, after which *A. fumigatus* could not be isolated. Half of the flasks were inoculated with a pure culture of the fungus isolated from a turkey lung. The fungus soon covered the surface of the mash. Unfortunately no turkey poults were available at this time. However, a lot of 17 one-day-old chicks was divided into three groups. Eight were fed the inocu-

lated mash; five were fed the sterile feed and four the original, non-autoclaved food. After eleven days, one of the chicks fed the inoculated material died. The lungs showed the typical acute lesions resulting from aspergillosis and the fungus was re-isolated from the tissues. The groups fed the sterile and the non-autoclaved mash remained normal. Possibly chicks are more resistant than turkey poults to aspergillus infection.

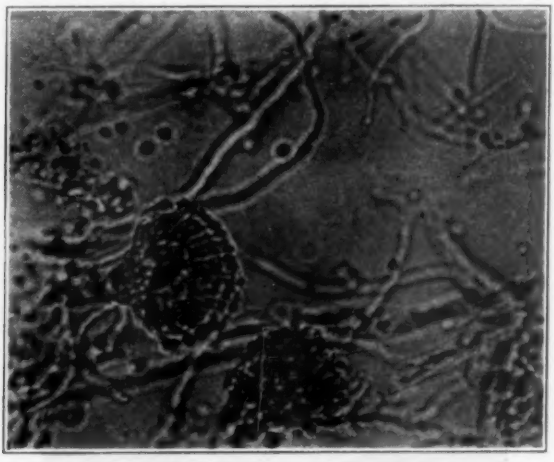

FIG. 3. Photomicrograph of *Aspergillus fumigatus* Fres, isolated from lung tissue of a wild turkey poult. Shows hyphae and a conidiophore with sterigmata and conidia.

The evidence indicates that *A. fumigatus* from infected feed was responsible for the epizoötic among the wild turkey poults.

The commercial feed contained cod-liver oil and fish meal and, under damp conditions, proved a good medium for the development of *A. fumigatus*. This report suggests the importance of using freshly prepared feeds, especially during the highly susceptible period of growth.

A SIMPLE DEVICE FOR PROTECTING WOUNDS OF THE HEAD IN DOGS*

By BENJAMIN MCINNES, *Charleston, S. C.*

Recently I read of a new device for restraining dogs from inflicting injuries about the head.†

Several years ago I was operating on a dog with a small tumor

*Received for publication, May 4, 1935.

†New restraining device. No. Amer. Vet., xvi (1935), 4, p. 45.

near the left eye. After finishing the operation, I said to my assistant that if I could prevent the dog from scratching the wound, it soon would heal. I thought about the problem for a few minutes with the following results: I devised a simple collar made of pressboard, about 3/16 of an inch thick, which may be made easily in a few minutes and which costs not more than five or ten cents. I saw the owner of the patient a few weeks after the operation and he told me that the collar was a perfect success.

Since the first trial of this simple device, we have used many of them. A collar will last for a couple of weeks and if, at the end of that time, another one is needed, the first one can be replaced with another at a very small cost.

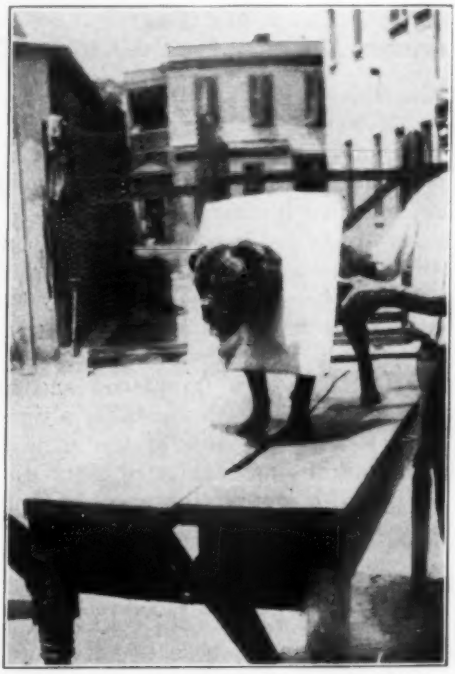

FIG. 1. Patient wearing pressboard collar.

The accompanying illustration (fig. 1) shows a dog wearing one of the collar-guards described in this article.

The pressboard has several advantages over other materials, including leather. In the first place, the veterinarian does not have to keep on hand collars of different sizes. Each one is

made to fit the patient and if the veterinarian follows this plan the collar always fits. Leather collars are difficult to cleanse thoroughly and when they get wet they bend and get out of shape easily. Furthermore, after a leather collar has been wet and dried, it is rather harsh on the neck and, being heavy, does not turn easily. If the dog scratches his head, the pressboard collar turns easily and thus prevents any abrasion of the neck. In making the collar, be careful to cut the hole perfectly round and smooth so that it will pass over the head readily. Do not make the guard too large. All that is necessary is to have it of such a diameter that the patient cannot reach the wound with one of the paws. Another consideration is that the device should not be large enough to prevent the animal from lying down comfortably.

Veterinary Hospital for the Chicago Zoo

Animals at the Chicago Zoo, at Brookfield, Ill., are soon to be provided with a $35,000 hospital, according to a recent announcement. A separate unit is planned to care for the sick birds. Included in the plans also are an autopsy room, a laboratory and an office for the veterinarian in charge.

Private cubicles are to be provided with air-conditioning. A maternity ward has been planned for the feminine residents, where they may have the same absolute privacy that they would have in the jungle. The new hospital is to be also a kind of quarantine station for the new mammal immigrants. Animals frequently arrive at the zoo in a frightened and highly nervous state. If they are kept in isolation wards for a time, they recover from their fright more easily and, at the same time, give the attending veterinarian an opportunity to check up on their general health. It is expected that the hospital will be completed within the next two or three months.

Dogs and Hose

A manufacturer of women's hose is advertising his product with pictures of dogs and suggestions of color combinations for the feminine wardrobe. With navy blue, the well dressed woman will wear cocker hose; with pastel shades, she will wear chow hose, and with prints, airedale.

There are 18 national game refuges and 181 national forests in the United States at the present time.

A GUIDE TO THE STUDY OF SPECIAL VETERINARY PATHOLOGY. R. A. Runnells, Associate Professor of Veterinary Pathology, Iowa State College. 218 pages. Collegiate Press, Inc., Ames, Iowa, 1935. Price, $3.

As a teacher of veterinary pathology I have long felt the need of an up-to-date textbook on pathology, dealing with the problem primarily from a veterinary angle. It consists of two parts: (1) Systemic Pathology and (2) Special Pathology of the Specific Infectious Diseases. General pathology is not included in the text. While the inclusion of this division of pathology would be desirable, the need for it is not so keen as is that for the divisions included in the book, since there are available a number of excellent texts of human pathology that can be adapted to courses in veterinary pathology in so far as general pathology is involved.

Most of the discussions are rather brief, as they would have to be in a work limited to this size, but they are well presented and include the most recent results of veterinary research. One who reads the book can not help but be impressed with the thoroughness with which the author has consulted the American and European literature in the preparation of it.

In order to keep down the size and cost of the book, no illustrations were included. This is unfortunate but the reason is obvious due to the conditions over which the author has no control. Obviously for the same reason, the discussion of functional disturbances associated with anatomical alterations is not a conspicuous feature of the book.

The author has included a bibliography at the end of each chapter and three additional pages of references to literature dealing with deficiency diseases, plant poisonings and disturbances of metabolism, at the end of the book, which add greatly to its usefulness. The appearance of this work should be most welcome to teachers and students of pathology. It will prove a valuable addition to the library of all those working with or interested in animal diseases.

The author is to be congratulated upon his attempt to fill a conspicuous gap in American veterinary literature. The writing

of a complete textbook of veterinary pathology, and keeping it up to date by bringing out frequent editions, is a task of such magnitude that it should occupy the major part of a lifetime. Furthermore, one who attempts such a task does so without any hope of financial reward from its sale. In fact, under the existing conditions, demand for a complete textbook of veterinary pathology is such that only a relatively small part of the cost of publication could be met from the sale of such a work. Therefore, we can not hope to have in our veterinary literature a textbook of pathology of the high degree of excellence to be found in human medical literature unless some source of subsidy can be found to bear a large proportion of the cost of publication. The quality of the text, which is the subject of this review, indicates that the author is equal to the task if in some way the cost of publication could be met.

E. T. H.

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Vitamin D in Caviar

Caviar may become a pleasant and epicurean substitute for cod-liver oil, following an experiment by Prof. M. Lepsky, of Soviet Russia, reported by *Science*. Twenty children suffering from rickets were fed caviar and, of this number, 17 were cured. Caviar is reported to be rich in vitamin D.

Teacher: "What cow in the United States is best known for the amount of milk it gives?"

Tommy: "Magnesia, ma'am; all the drug stores sell its milk."

Truth is generally the best vindication against slander.—ABRAHAM LINCOLN.

EXPERIMENTAL RABIES IN WHITE MICE AND ATTEMPTED CHEMOTHERAPY. Anson Hoyt, Roy T. Fisk and Clinton H. Thienes, Jour. Inf. Dis., lvi (1935), 1, p. 21.

Groups of white mice, each comprising approximately 24 animals, were given intracerebral injections of fixed rabies virus. They were subsequently treated with various drugs in an attempt to prevent or modify the course of the disease. The drugs employed were plasmochin, merthiolate, metaphen, bismuth violet, iodobismitol, bismaren, tryparsamide, silver arsphenamine, Bayer 205, ethylhydrocupreine hydrochloride (optochin), pyridium, sodium arsanilate (atoxyl), and sparteine sulfate. The virus and the drugs were injected in amounts directly proportional to the body weight of each mouse. The experiments were divided into 16 series and control groups of mice were instituted in each series. The mean period of incubation and mean length of life did not differ significantly as between the treated and control groups of mice included in any one series of experiments.

TULAREMIA IN WILD GRAY FOXES: REPORT OF AN EPIZOÖTIC. Carl F. Schlotthauer, Luther Thompson and Carl Olson, Jr. Jour. Inf. Dis., lvi (1935), 1, p. 28.

Tularemia was found in ten wild gray foxes; seven animals were found dead while three were found sick. The animals were emaciated, had diarrhea and were heavily infested with fleas and some ticks (*Ixodes cookei*). Congested visceral lymph-nodes and tubercle-like lesions in the lungs were constant lesions in all foxes. Miliary white foci occurred on the livers and spleens of two animals. There were no gross lesions on the heart or kidneys. Histologically the lesions resembled those of tuberculosis. They consisted of endothelial cells and fibroblasts, with infiltration by lymphocytes, large monocytes and polymorphonuclear leukocytes. *Pasteurella tularensis* was isolated from four of the animals. A limited number of transmission experiments to guinea pigs and rabbits were positive for these animals.

THE PASSAGE OF BACTERIA FROM THE LUNGS INTO THE BLOOD-STREAM. William M. Tuttle and Paul R. Cannon. Jour. Inf. Dis., lvi (1935), 1, p. 31.

Bacterial suspensions of *Staphylococcus aureus, Bacillus prodigiosus* and *Streptococcus hemolyticus* were introduced into the left lower lobe of the lungs in 14 healthy dogs and samples of blood from the femoral artery and lymph from the thoracic duct were cultured at frequent intervals during the first 60 minutes. In animals injected with staphylococci and *B. prodigiosus*, both the blood and lymph remained sterile throughout the period of observation. The bacteria were demonstrable in the lungs at the end of the experiment. In the case of hemolytic streptococci the microörganisms quickly entered the blood-stream, whereas the thoracic lymph-duct remained sterile, proving that the passage of the bacteria was not through the lymphatics. The differences in response of the pulmonary tissues to the microörganisms employed seemed to depend upon the varying degrees of injury to the cellular and tissue barriers within the lungs and that experimental bacteremia occurred because of an increased permeability of these membranes.

THE INFLUENCE OF BOVINE SERUM ON BRUCELLA INFECTION IN GUINEA PIGS. B. A. Beach. Jour. Inf. Dis., lvi (1935), 1, p. 38.

The administration of bovine serum showing germicidal power to guinea pigs, either subcutaneously or intra-abdominally, retarded Brucella infection as compared with the controls. The author concludes that unless some method is found of increasing the protective power of cow serum it will probably be of little practical value in combating Brucella infections.

THE SPONTANEOUS OCCURRENCE OF BRUCELLA AGGLUTININS IN DOGS. William H. Feldman, Frank C. Mann and Carl Olson, Jr. Jour. Inf. Dis., lvi (1935), 1, p. 55.

Brucella agglutinins were found to be present in the serums of 52 (10.4 per cent) of 500 adult dogs from rural environments. Serums from 15 of the reactors failed to agglutinate the antigens in dilutions greater than 1:12, whereas serums of 33 (6.6 per cent) of the animals were positive in dilutions of 1:25 or greater. Serums of only five animals were positive to the agglutination test in titres of 1:100 or greater. Negative results were obtained in attempts to isolate *Brucella abortus* by means of guinea

pig inoculation with emulsions of tissues from 14 of the reacting animals. Dissemination of the infection from dogs with agglutinins against Brucella by cohabitation with normal dogs failed. None of the dogs with agglutinins against Brucella exhibited symptoms indicative of specific infection. Necropsy failed to reveal pathologic evidence of disease due to *Br. abortus*. Experiments to determine cross-agglutinability of *Br. abortus* and *Bacillus bronchisepticus* in dogs and rabbits yielded negative results.

THE PATHOGENICITY FOR CATTLE OF BRUCELLA STRAINS ISOLATED FROM CASES OF UNDULANT FEVER IN MAN. R. R. Birch and H. L. Gilman. Jour. Inf. Dis., lvi (1935), 1, p. 78.

Brucella strains isolated from typical cases of undulant fever in man proved regularly to be pathogenic for cattle, producing in them a syndrome indistinguishable from that which occurs in natural cases of Brucella infection otherwise known as infectious abortion.

THE BACTERICIDAL PROPERTIES OF ADRENAL EXTRACTS. J. Gordon and J. C. Knox. Abst. Arch. Path., xix (1935), 3, p. 428.

The results described indicate that after prolonged extraction of the bovine suprarenal gland with water at 65° C. without exclusion of air, a substance is present in the dark-colored extract, which has a marked inhibitory effect on bacterial growth. This inhibition is most evident with *Bacillus diphtheriae* and *Vibrio cholerae*, but there is a wide range of other bacteria which show this inhibition in greater or lesser degree.

THE LESIONS PRODUCED IN THE UTERUS OF THE GUINEA PIG BY INJECTIONS OF SUSPENSIONS OF HEMOLYTIC STREPTOCOCCI AND OF THE PYOCYANEUS BACILLUS AT DIFFERENT STAGES OF THE SEXUAL CYCLE. C. S. Linton, A. A. Kippen and Leo Loeb. Jour. Inf. Dis., lvi (1935), 1, p. 89.

Fifteen guinea pigs, which were injected (right horn of uterus injected, left horn as control) one day after estrus showed a marked reaction. Of two guinea pigs injected three days after estrus, one animal showed a marked reaction while the reaction in the other was less marked, the leukocyte reaction being absent. Seventeen guinea pigs were injected four and five days following estrus, showed a slight reaction. There were no leukocytic accumulations except near the wound. Of twelve

guinea pigs which were injected eight and ten days after estrus, seven showed a slight reaction while five showed a more marked reaction. Of six guinea pigs injected eleven and twelve days after estrus, five showed a marked reaction. The leukocytic response was more pronounced after injection of *B. pyocyaneus* than streptococcus.

INTRAPERITONEAL INJECTION OF RABIES VIRUS. A. C. Marie. Abst. Arch. Path., xix (1935), 3, p. 430.

The injection of Chinese ink subcutaneously or into the bloodstream of guinea pigs had been shown previously by the author sometimes to facilitate and sometimes to prevent rabies when the virus was injected intramuscularly. Intraperitoneal injection of Chinese ink, however, reduced the resistance of the serous surfaces of the peritoneal cavity by blocking the reticulo-endothelial system to such a point that infection with a fixed virus took place. Van Deinse's mixture of disodium phosphate and calcium chloride was also successful for blocking. The results were clear cut, and the controls confirmed observations that intraperitoneal injections of rabies virus rarely result in infection.

PRODUCTION AND COMPOSITION OF SOW'S MILK. E. H. Hughes and Hugh G. Hart. Jour. Nutri., ix (1935), 3, p. 311.

Sows produce about 6.8 pounds of milk per day for an average lactation period of 8 to 12 weeks. The average for the constituents of colostrum as determined in these studies were: total solids 28.02, fat 5.96, protein 15.49 and ash 0.65 per cent. The averages for normal milk were: total solids 17.98, fat 6.77, protein 6.22 and ash 0.97 per cent. The average calcium and phosphorus content of colostrum of sow's milk were 0.076 and 0.083 per cent, respectively; while for normal milk they were calcium 0.252 and phosphorus 0.151 per cent. This latter calcium-phosphorus content is higher than that reported for the human or the cow. The ash content of sow's milk increases during lactation.

STUDIES ON BLOOD COAGULATION. I. The rôle of prothrombin and of platelets in the formation of thrombin. Harry Eagle. Jour. Gen. Physiol., xviii (1935), 4, p. 531.

Thrombin cannot be considered as an artificial by-product of coagulation. Calcium, a platelet factor and a plasma factor (prothrombin) interact to form thrombin and this acts upon

fibrinogen to form fibrin. The amount and rate of thrombin formation are independent of the presence or absence of fibrinogen. The amount of thrombin formed in a mixture of prothrombin, calcium and platelets is independent of the platelet or calcium concentration and depends primarily upon the amount of prothrombin used. The platelets enormously accelerate the transformation of prothrombin to thrombin. No evidence is available to determine whether the platelet factor actually combines with prothrombin to form thrombin or merely catalyzes the transformation.

STUDIES ON BLOOD COAGULATION. II. The formation of fibrin from thrombin and fibrinogen. Harry Eagle. Jour. Gen. Physiol., xviii (1935), 4, p. 547.

Although calcium is essential for the formation of thrombin, it can be recovered from formed horse thrombin without affecting its coagulating activity. This is evidence that fibrin is not a calcium-protein compound. The weight of evidence favors the theory that thrombin is an enzyme analogous to rennin and not that it combines with fibrinogen to form an insoluble fibrin. A given quantity of thrombin can form at least 200 times its weight of fibrin.

INFLUENCE OF LOCAL INFLAMMATION OF TETANUS AND OTHER PROCESSES. M. Galea and E. Falchetti. Abst. Arch. Path., xix (1935), 3, p. 428.

Inflamed lesions were invoked in guinea pigs by avirulent tubercle bacilli or by grain infusions. Tetanus spores injected into these lesions spread more rapidly than in normal animals. The spread was more rapid in the lesions caused by tubercle bacilli, but such areas were less favorable for spore germination. Lesions induced by the toxin of *Clostridium oedematiens* were rapid but less serious in animals with foci of inflammation than in controls, the lesions favoring the elimination of the toxin. The virus of foot-and-mouth disease was retained in the skin, especially in lesions induced by *BCG*, longer than in the blood or the internal organs, but inflammatory processes did not affect the course of the infection. The authors see in these results a defense mechanism analogous to that occurring in vaccination with *BCG*.

National Dog Week this year will be observed September 22-28. The slogan for the Week is, "Every Home Needs a Dog."

Regular Army

Major Raymond Randall is assigned to duty at the Boston quartermaster depot, Boston, Mass., effective upon completion of his present tour of foreign service in the Panama Canal Department.

So much of special orders as assigns Major Ralph B. Stewart to duty at Fort Des Moines, Iowa, upon completion of his tour of foreign service in the Philippines, is amended so as to assign him to duty at Fort Riley, Kan.

So much of par. 59, S. O. 60, W. D., 1935, as relates to Major Kenneth E. Buffin is revoked. Major Kenneth E. Buffin is relieved from further assignment and duty at Fort Sam Houston, Texas, effective in time to comply with this order, will then proceed to Carlisle Barracks, Pa., and report to the commanding general not later than May 15, 1935, for duty.

Veterinary Reserve Corps

New Acceptances

Hamman, Fred Irwin..... 1st Lt...P. O. Box 216, Loveland, Colo.
Jones, Marion James..... 2nd Lt...Box 481, Milaca, Minn.
Rash, Edd Ellis..... 2nd Lt..302 Federal Bldg., Cheyenne, Wyo.
Smit, Charles Rudolph..... 2nd Lt...Alton, Iowa.

Promotion

To

Catlett, James Garland..... Lt. Col...2418 N. Miami Ave., Miami, Fla.

Active Duty for Veterinary Reserve Corps Officers

Reserve veterinarians desiring active service with the Civilian Conservation Corps for a period of six months, or longer, should apply immediately to the commanding general of the Corps Area in which they reside for active duty.

Army Legislation

A more efficient system of promotion for officers in the U. S. Army is that proposed by Senator Morris Sheppard, of Texas, in Bill S. 1404, which was read before the Senate on March 29, 1935, passed by that group and which is now being considered by the House Military Committee.

Concerning that part of the present law that deals with the Veterinary Corps, Senator Sheppard spoke as follows:

The proposed law gives veterinary officers and chaplains the same system of appointment and promotion as is now given officers of the Medical Corps. At present, officers of the Veterinary Corps are appointed in the grade of second lieutenant, are promoted to first lieutenant after three years' service, to captain after seven years' service, to major after 14 years' service, to lieutenant colonel after 20 years' service, and to colonel after 26 years' service.

Chaplains now are appointed as first lieutenants, reach captain in five years' service, major in 14 years, lieutenant colonel in 20 years, and get no higher.

Officers of the Medical Corps are appointed as first lieutenants, promoted to captain after three years' service, to major after 12 years, to lieutenant colonel after 20 years, and to colonel after 26 years.

Senator Sheppard then went on to explain the advantages the new law would have over that now in effect.

Chaplains and veterinary officers are placed on a parity by this bill with officers of the Medical Corps for the reason that it is desired from the standpoint of simplified administration to include the bulk of non-promotion-list officers in the same system of promotion, and at the same time to accord an appropriate and a too-long-delayed recognition to two important branches of the Army.

Section 4 further provides that from and after the effective date of this act original appointments in the Veterinary Corps shall be made in the grade of first lieutenant from Reserve Veterinary officers between the ages of 23 and 32 years, and that officers serving in that Corps on the effective date of this act in the grade of second lieutenant shall be promoted to the grade of first lieutenant as of said date.

At present initial appointments to the Veterinary Corps are made in the grade of second lieutenant, whereas officers of the Medical Corps and Chaplain Corps are initially appointed first lieutenants. As explained, just previously, the first part of section 4 is to equalize the length of service required for promotion in the three branches wherein officers are promoted after certain years of service, namely, Medical Corps, Veterinary Corps and Chaplain Corps. This last clause of section 4 provides that veterinary officers will also enter the service in the same grade as do medical officers and chaplains.

It was the contention of Senator Sheppard that, in the grade below colonel where promotion is by seniority, officers should reach each grade at such an age as to render the best possible service for that particular status and should not continue in the same grade too long after having mastered its possibilities and requirements, or too long to reach the next grade at an age early enough to do it full justice, or so long as to upset the age balance on which a promotion system should be based. The chief object of a promotion system is to secure the maximum benefit to the service, Senator Sheppard emphasized. In the promotion of officers, the principal purpose should be to obtain the best commanding personnel.

COMMENCEMENT

ONTARIO VETERINARY COLLEGE

Forty candidates received the degree of Bachelor of Veterinary Science at the Convocation of the University of Toronto, May 10, 1935. They are:

A. O. Beach
G. E. Bitgood, Jr.
J. E. Blake
A. Bodendistel
E. J. Burke
H. J. Cairns
W. B. Cameron
J. P. Cassidy
W. B. Coxon
W. G. Davidson
D. L. Diamond
T. R. Eliason
J. F. Evans
D. J. Faragher
L. H. Ferris
A. E. Freeman
H. Gough
H. H. Habel, Jr.
Thomas Harper
V. M. Hensler
Wm. J. Hoey
G. A. Jelly
J. M. Kaine
Fred Keefe
B. L. Lawlor
B. F. Leach
Fred J. Leslie
M. P. Maher
A. E. McEwen
C. L. McGilvray
R. L. Mummery
G. E. Myers
P. J. Pascoe
E. M. Powers
F. J. Powley
J. W. Sutherland
R. C. Topp
P. R. Thorne
R. I. Twiss
A. H. Watt

Honors were awarded to members of the graduating class as follows:

Honor Standing

Gold Medal for highest aggregate standing—Thomas Harper, of North Shields, England.

Second Prize—B. F. Leach, of East Fairfield, Vermont.

Third Prize—L. H. Ferris, of Flesherton, Ont.

Andrew Smith Memorial Medal

W. G. Davidson, of Toronto, Ont.

Helen Duncan McGilvray Prize

B. F. Leach, of East Fairfield, Vt.

Bacteriology, Special Prize

Thomas Harper, of North Shields, England

Canadian Army Veterinary Corps Prize

Wm. J. Hoey, of Vancouver, B. C.

Clinical Practice Prize

R. C. Topp, of Smithville, Ont.

Science Association Prizes

Thomas Harper, of North Shields, England
C. L. McGilvray, of Guelph, Ont.

K. S. C. Student-Alumni-Faculty Dinner

Students in the Division of Veterinary Medicine at Kansas State College held their own Recognition Day ceremonies on the occasion of the annual student-alumni-faculty dinner, April 27, 1935, instead of waiting for the annual twelfth Recognition Day exercises, on May 3, when awards were made to honor students in all divisions of the College.

The affair was sponsored by the Student Chapter of the American Veterinary Medical Association. The banquet was held in the Masonic Temple, Manhattan, with about 250 students, members of the faculty and alumni in attendance. Dr. Louise Sklar (K. S. C. '34) was the faculty supervisor in charge of arrangements. Mr. Clarence Schmidt, senior student in veterinary medicine, acted as toastmaster. Responses to toasts were made by Dr. W. E. Grimes, head of the Department of Agricultural Economics, and Dr. E. L. Holton, dean of the Summer School. Both addresses received the closest attention, and were among the best that have been given on similar occasions.

Prizes and Certificates of Merit to honor students in the Division of Veterinary Medicine were awarded at the banquet, this part of the evening program varying from that of previous years. The following cash awards, in each case accompanied by a Certificate of Merit, were given:

General Proficiency

Prizes offered by Dr. E. A. Schmoker (K. S. C. '17), of Seattle, Wash.
First Prize ($10).....Oscar Frederick Fisher, '35
Second Prize ($5).....Donald Clifford Kelley, '35

Pathology

Prizes offered by Dr. O. M. Franklin (K. S. C. '12), of Amarillo, Tex.
First Prize ($10).....Joseph F. Knappenberger, '35
Second Prize ($5).....Eusebio Antonio Perez Herrera, '35

Physiology

Prizes offered by Dr. N. D. Harwood (K. S. C. '18), of Manhattan, Kan.
First Prize ($10).....Lee Thomas Railsback, '37

KANSAS STATE COLLEGE VETERINARY GROUP

Back row: Dr. Herman Farley, Dr. J. H. Burt, Dr. J. P. Scott, Dr. R. R. Dykstra (Dean), Dr. E. J. Frick, Dr. C. A. Brandly (Department of Bacteriology).

Middle row: Dr. W. M. McLeod, Dr. J. H. Whitlock (Department of Zoölogy), Dr. E. R. Frank, Dr. H. F. Lienhardt, Dr. Glen L. Dunlap (U. S. B. A. I.), Dr. C. H. Kitselman.

Front row: Dr. E. E. Leasure, Dr. Louise Sklar, Dr. Helen S. Richt, Dr. Gordon Danks, Dr. C. L. Adan.

Dr. C. A. Pyle, anaplasmosis research agent, at Sedan, Kan., and Dr. J. W. Lumb, extension veterinarian, are not included in the picture.

*Second Prize ($5) ..... {Guy William Bayles, '37
Willard Merrill Van Sant, '37

Small-Animal Clinic

Prizes offered by Dr. C. W. Bower (K. S. C. '18), of Topeka, Kan.
First Prize ($10) ..... Lawrence Charles Donat, '35
Second Prize ($5) ..... Harold Jack Jewell, '35

Therapeutics

Prizes offered by Dr. C. E. Salsbery (K. C. V. C. '11), of Kansas City, Mo.
First Prize ($10) ..... Loris Arthur Dehner, '36
Second Prize ($5) ..... Edgar William Millenbruck, '36

Veterinary Article

Prizes offered by Dr. D. M. Campbell (K. C. V. C. '07), editor of *Veterinary Medicine*, Chicago, Ill.
First Prize ($10) ..... Hubert Raymond Hein, '35
Second Prize ($5) ..... Edwin Strand Wiseman, '35

Honorable Mention:

Edwin Louis Millenbruck, '35
Leonard Anthony Rosner, '35
Jacob Emil Spring, '35
Charles D. Stafford, '35
Clarence M. Stay, '35
Theodore S. Williams, '35

After the banquet, the group gathered in the ballroom of the Wareham Hotel for dancing.

The election of the following veterinary students to Gamma Sigma Delta, honorary society in agriculture and related sciences, was announced at the regular Recognition Day exercises on May 3: Joseph L. Cavanaugh, Lawrence C. Donat, Herbert H. Fechner, Oscar F. Fischer, Donald C. Kelley, Joseph F. Knappenberger, Edgar W. Millenbruck and Theodore S. Williams.

New York Selects Veterinarian for Important Post

Dr. A. L. Brown (Corn. '15), of Deposit, N. Y., became Assistant Commissioner of Agriculture and Markets for New York State, on May 1, 1935, succeeding Mr. Christian P. Norgord, whose retirement was effective on that date. Two weeks later, Mr. Peter G. Ten Eyck, Commissioner of Agriculture and Markets, promoted Dr. Brown to the office of Director of the New York State Fair, with headquarters at Syracuse. The appointment of Dr. Brown to this high position represents a definite move on the part of Commissioner Ten Eyck to keep in close contact with the Fair management and to make the Fair of outstanding service to the State.

*Prize divided.

Dr. Brown is well fitted for the duties of his new office. After eleven years spent in general practice and inspection work, he became veterinarian for the Borden Farm Products Company, Inc., and was promoted to District Veterinarian. His work in this position took him over the whole of New York State and parts of other states and brought him in close contact with boards of health, county agricultural agents, farm bureaus and other groups dealing with agricultural problems. He was elected to the State Assembly in 1932, defeating the veteran Republican, Edmund B. Jenks.

DR. A. L. BROWN

Dr. Brown has always been active in civic affairs. He served as president of the Deposit Chamber of Commerce, and was for two terms a member of the Board of Trustees of the village, serving one term as president. He is at present a member of the Board of Education of Deposit and is an active student of rural school problems. Dr. Brown was born near Carthage, Jefferson County, N. Y., and at one time operated a fruit farm at Oswego.

Another Abortion Remedy Banned by Court

E. C. Bellwood, of South Richmond, Va., manufacturer of Bellwood Farms Abortion Treatment, has been found guilty of violating the Federal Food and Drugs Act by shipping his so-called remedy for Bang's disease in interstate commerce. The Federal Court, at Richmond, has placed Bellwood on probation for three

years, during which time he is obligated not to violate the Food and Drugs Act or other criminal laws of the United States. If he does illegally ship his nostrum in interstate commerce, the Court may pronounce sentence upon him. The fraudulent concoction, sold by Bellwood, trading as Bellwood Farms, was found to consist of cornstarch with just enough potassium permanganate to make a pink solution in water. The ingredients used cost but a few cents a pound, but the farmers who used the treatment paid from $6 to $12 for a package weighing between a pound and a pound and a half.

Departments Merged at Virginia Polytechnic Institute

A little less than ten years ago—in the December, 1925, issue to be exact—the JOURNAL carried a news item to the effect that the name of the Department of Veterinary Science of the Virginia Polytechnic Institute had been changed to the Department of Zoölogy and Animal Pathology, through the addition of zoölogy to the Department. Dr. I. D. Wilson (Iowa '14) continued in charge.

DR. I. D. WILSON

This issue of the JOURNAL records another change. The Department of Botany and Plant Pathology has been combined with the Department of Zoölogy and Animal Pathology, and will function as the Department of Biology under the direction of Dr. Wilson. The work of the new department now consists of botany, plant pathology, bacteriology, entomology and animal pathology.

The Lowdown on Oklahoma Weather

Oklahoma City may sound as if it might be beset by hot weather in August. On the contrary, it is cooler than a great many other cities in which the A. V. M. A. has met in the past, according to official figures from the U. S. Weather Bureau. A report covering the mean maximum and minimum temperatures at Oklahoma City for the last week in August during the past ten years reveals the following data:

MEAN TEMPERATURES

Year	*Maximum*	*Minimum*
1925	93.9	69.9
1926	85.9	65.4
1927	83.1	66.1
1928	91.3	69.7
1929	95.0	70.4
1930	90.9	67.7
1931	92.9	68.4
1932	91.9	72.0
1933	81.4	66.1
1934	86.7	67.1
Average	89.3	68.3

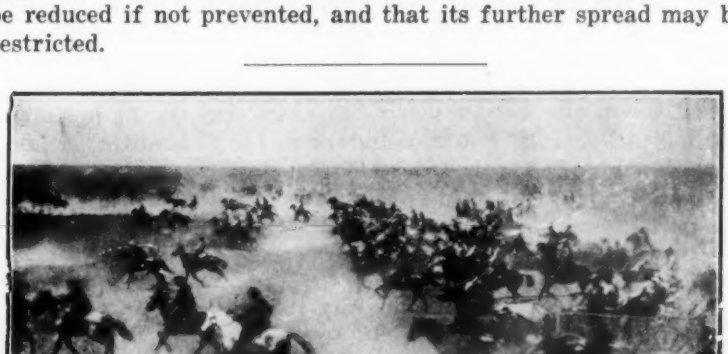
For those who may be skeptical, there are the figures. According to Dr. C. H. Fauks, secretary of the Committee on Local Arrangements, the outlook for pleasant daytime weather for the A. V. M. A. meeting is decidedly promising, to say nothing of the cool breezes that are common to the nighttime.

Elected to Sigma Xi

Three eminent scientists—Dr. John R. Mohler, Dr. S. F. Blake and Dr. O. S. Adams—were initiated into the Washington, D. C., Chapter of Sigma Xi, national honorary scientific society, at the annual banquet held at the University Club, May 14, 1935. Acknowledging the honor bestowed upon them, the three new members responded with talks on their specialties. Dr. Mohler, Chief of the U. S. Bureau of Animal Industry, discussed "Explorations Beyond the Microscope." He pointed out that scientific study of the ultraviruses, which are disease producers so minute that they cannot be seen even with the most powerful microscope, must be done chiefly by observing the phenomena they produce. Such observations, however, have resulted in the production of serums and other means of preventing or controlling a number of important live stock diseases. Dr. Blake, of the U. S. Bureau of Plant Industry, spoke on the Compositae, the largest single plant family. Dr. Adams, of the U. S. Coast and Geodetic Survey, told of some mathematical aids in map projections.

War Declared Against Screw Worm

A battle against the screw worm is now in progress in the southern states. The U. S. Department of Agriculture, with a Congressional appropriation of $480,000, has joined forces with Florida, Georgia, Alabama, Mississippi, Louisiana, South Carolina and southeastern Texas to eradicate the pest. Part of the fund will be used to send out agents to show groups of farmers and ranchers how to protect stock against the screw worm. Another part will buy benzol and pine tar oil for treating infested animals, as well as material for building pens and chutes in which animals are held during treatment. A third part will be used for research to develop cheaper and more effective control measures. It is the hope of the Department of Agriculture that, in coöperation with state and local agencies, the losses from the huge worm army, now over-wintering in the southern area, may be reduced if not prevented, and that its further spread may be restricted.



"The Run," Marking the Opening of Oklahoma and the Founding of Oklahoma City.

Oklahoma Veterinary Roster

A roster of the graduate veterinarians in Oklahoma, prepared and distributed by the Oklahoma Veterinary Medical Association, shows a majority of the 168 members listed as members of the A. V. M. A., the state association, or both. Of the 136 practitioners listed, 79 (58 per cent) are members of the A. V. M. A., and 94 (69 per cent) are members of the Oklahoma Veterinary Medical Association.

The personnel of the U. S. Bureau of Animal Industry, which includes eight members on the field force, ten on the meat-inspection force and two on the virus-serum control force, boasts a 100 per cent membership in the A. V. M. A. All members of the field force and two members of the meat-inspection force belong to the state association.

The roster includes two army veterinarians, both A. V. M. A. members, four veterinarians at the Oklahoma A. & M. College, who are members of both the national and state associations, and six veterinarians on the state control force, three of whom are members of both organizations. Of the 168 veterinarians included in all classifications, 46 per cent are affiliated with the A. V. M. A., and 66 per cent with the Oklahoma Veterinary Medical Association.

Dr. C. H. Fauks, secretary of the state association, directed the publication of the roster.

To Study Bighead in Sheep

Dr. W. T. Huffman (Gr. Rap. '08), of the U. S. Bureau of Animal Industry, Salt Lake City, Utah, will assist in making studies of the causes of bighead in sheep, which are to be carried on this summer under the supervision of Arthur B. Clawson, of the U. S. Department of Agriculture. Other assistants to Mr. Clawson are E. A. Moran, of the U. S. Bureau of Animal Industry, and Milton A. Madsen, of Manti, Utah. The studies will be carried out on the live stock range in Utah, Idaho and neighboring states, and will attempt to prove that bighead is caused by coal oil weed or spineless horsebrush.

Annual Meeting of Phi Zeta at O. S. U.

The annual initiation ceremony and banquet of Delta Chapter of Phi Zeta, national veterinary honor society, was held at the Faculty Club of Ohio State University, May 9, 1935.

Two honorary members and 14 active members were received into the society and were guests of honor at the banquet that preceded the initiation. Those admitted to honorary membership were: Dr. Carl W. Gay (Corn. '99), chairman of the Department of Animal Husbandry, Ohio State University, and Dr. Stanton Youngberg (O. S. U. '07), former Director of the Bureau of Animal Industry, Philippine Department of Agriculture. Six members of the senior class of the College of Veterinary

Medicine were elected to active membership: Claude A. Smith, Harold M. Spangler, Charles M. Parker, Dwight G. Herd, Charles E. Haupert and Joseph E. Badger. Members of the junior class elected were: Fred Allen, V. W. Zuercher, G. A. Dick, R. P. Wagers, W. E. Bills and Robert Dougherty.

Dean O. V. Brumley, president of Delta Chapter, presided as toastmaster at the banquet. Dr. Youngberg gave an address in which he emphasized the relationship between the veterinary profession and the economic development of the Philippine Islands.

R. E. R.

Horse and Mule Pulling Contest

A horse and mule pulling contest, sponsored by the *Chicago Tribune*, will be held on June 15, 1935, at the Tribune Auxiliary Experimental Farm, two miles west of Wheaton, Ill. Fourteen cash prizes will be awarded the winners. There will be no entry fee. The contest is open to horses of every breed and color, and to mules of any type or color or of any age. It is being held with the coöperation of the Horse and Mule Association of America, and will be conducted under the rules and regulations of the Association. Mr. Wayne Dinsmore, secretary of the Association, is in charge of the technical details.

The purposes of the contest are: (1) To aid farmers and city team-owners in recognizing and developing economic horse and mule power through better breeding, feeding, conditioning, training and driving. (2) To demonstrate the value of proper harnessing and efficient driving. (3) To assist in building good will and to aid in a better understanding between city and country.

Entry blanks, which will be supplied by the *Chicago Tribune*, must be mailed not later than June 8.

Veterinary Exhibit at San Diego Exposition

The veterinary exhibit at the California Pacific International Exposition, San Diego, represents a coöperative undertaking on the part of veterinary associations, veterinary colleges, veterinary supply houses, and the Mayo Foundation of Rochester, Minn. The Exposition opened on May 29 and will last through November 11, 1935. The exhibit, which occupies a floor space of 18 by 34 feet, is in the Palace of Science and has been designed to present a graphic picture of the various activities of the vet-

erinarian. The task of assembling and arranging the exhibit was largely in the hands of the members of the San Diego County Veterinary Medical Association, working through a Committee on Arrangements consisting of Drs. C. R. Schroeder and F. G. Ward.

Members of the California State Veterinary Medical Association will convene at San Diego for the summer meeting beginning June 17. The last day of the meeting, June 19, has been designated as "Veterinarians' Day." The Exposition is offering its facilities for the occasion, and the state association is making plans for an interesting series of events throughout the day. Dr. H. L. Simpson, of Escondido, is chairman of the committee for the special day. The JOURNAL will publish further details concerning the exhibit just as soon as these are available.

Veterinarian to Administer Dairy Products Act

The provincial government of New Brunswick, Canada, has appointed Dr. L. A. Donovan (Ont. '16), of Saint John, N. B., chairman of the recently appointed commission to administer the New Brunswick Dairy Products Act, which was passed last March. The commission, headed by Dr. Donovan, holds wide powers over the distribution of dairy products in New Brunswick.

In addition to his new office, Dr. Donovan is director of the Saint John Exhibition Association and chairman of its live stock committee, a director of the Amherst Winter Fair, president of the New Brunswick Ayrshire Breeders' Club and past maritime director of the Ayrshire Breeders' Association. With his brothers he owns the famous Donovan Ayrshire herd at Glen Adler Farm, Coldbrook, which was started by his grandfather, Lawrence Donovan.

T. F. J.

Legislation to Protect the American Eagle

The American or bald eagle, which was adopted as the national bird by Act of the Continental Congress on June 20, 1782, is in danger of extermination unless legal steps are taken at once to protect it. Such steps are now being taken through the medium of House Bill No. HR 5271 which has the backing of the National Audubon Association.

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The April meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker on the 3rd. The speaker of the evening was Dr. Ervin A. Tusak, junior ophthalmological surgeon at Memorial Eye Hospital, and chief of the out-patient clinic, Bellevue Hospital, New York City. Dr. Tusak continued with his discussion of the diseases of the eye, the first part of which he presented at the March, 1934, meeting of the Association.* This lecture was confined to a discussion of the iris from the medical and surgical aspects. Diagnosis and treatment of diseased conditions of the iris were carefully outlined. Dr. A. Eichhorn, of Pearl River, N. Y., showed two films from the library of Davis & Geck which portrayed the surgical technic for nephrotomy and thoracoplasty.

May Meeting

The May meeting was held at the College of Pharmacy of Columbia University on the evening of the 1st. This session was devoted to a clinic, conducted by Dr. E. F. Schroeder, of the Angell Memorial Animal Hospital, Boston, Mass. Dr. Schroeder first spoke on "The Handling of Fractures," illustrating his lecture with lantern-slides. Several of the members present produced dogs with severe fractures, and Dr. Schroeder, with the skill of a magician, proceeded to apply his modified Thomas splint and rectify the damage to the shattered bones. X-ray pictures were taken of the injured parts both before and after the splints were applied. Dr. Schroeder's demonstration won loud acclaim from an enthusiastic membership. The x-ray machine used in the demonstration was lent by the General Electric Company. Through the courtesy of the technicians supplied by the Company, the pictures were taken and developed immediately, so that the practitioners might fully appreciate the splendid demonstration presented by Dr. Schroeder.

R. S. MacKellar, Jr., *Secretary.*

*Tusak, E. A.: Surgical and medical procedure in the treatment of external diseases of the eye in veterinary practice. Jour. A. V. M. A., lxxxv (1934), n. s. 38 (3), pp. 379-388.

NEW MEXICO VETERINARY MEDICAL ASSOCIATION

The fourth annual meeting of the New Mexico Veterinary Medical Association was held at the Franciscan Hotel, Albuquerque, May 4, 1935. Owing to the general economic situation, the third annual meeting, which should have been held in 1934, was omitted.

Programs distributed at the meeting were unusually designed, the color and typography of the cover carrying out the Navajo motif. In addition to the regulation announcements, the program included also a well presented history of the Association, entitled "Up to Now," describing the activities of the profession in New Mexico from the founding of the New Mexico Veterinary Medical Association on January 15, 1931, to the present time. Following the opening address given by the President, Dr. F. H. Barr, of Albuquerque, the meeting for the day was taken up in part with the following papers:

"Common Internal Parasites of Sheep Encountered on Postmortem Examinations in the Field," Dr. H. E. Kemper, U. S. B. A. I., Albuquerque.

"Typhus in Dogs and Enteritis in Cats," Dr. T. I. Means, Santa Fe.

"Foreign Bodies in Cows and Dogs," Dr. H. C. Schipman, Las Cruces.

"Common Skin Ailments in Dogs," Dr. S. W. Wiest, U. S. B. A. I., Santa Fe.

In the afternoon, a clinic was held at the hospital of Dr. W. F. Fish, and was conducted by Dr. Harry Koll, of El Paso, Tex. Dr. Koll's demonstration of advanced clinical procedure was greatly appreciated. The annual banquet was held at the Franciscan Hotel in the evening.

Officers chosen to serve during the coming year are: President, Dr. F. L. Schneider, Albuquerque; vice-president, Dr. V. H. Magatagan, Albuquerque; secretary-treasurer, Dr. T. I. Means, Santa Fe. Steps to affiliate with the A. V. M. A. were taken at this meeting.

T. I. MEANS, *Secretary.*

HUDSON VALLEY VETERINARY MEDICAL SOCIETY

The regular quarterly meeting of the Hudson Valley Veterinary Medical Society was held at the U. S. Military Academy, West Point, N. Y., May 9, 1935. After luncheon at the Hotel Thayer, which was attended by more than 100 members and guests, the meeting was called to order by the President, Dr.

Thomas Sheldon, of Rhinebeck, N. Y., and a brief business session was held.

Members were then taken to the spacious kitchens of the Academy, where an unusually complete meat and food demonstration had been prepared. Majors E. M. Curley, Station Veterinarian, H. K. Moore, of the Army Base at Brooklyn, and H. E. Van Tuyl, of Fort Hamilton, explained the various steps taken in examining these foods before their acceptance for official use. Beef carcasses showing the different grades were available for comparison, and the system of determining such grades was explained by the experts. The grading of poultry, eggs and other food products was described also. A large array of processed meats and canned goods of various kinds were on display, and methods of examination were explained. The thorough and painstaking manner in which these foods are now being examined before they are accepted by the army veterinary officers impressed upon all the safeguards that are taken to prevent any repetition of the "embalmed beef" scandals of the past. After the demonstration, several questions were asked by interested listeners, and Col. J. R. Underwood, of the Army Medical Center, Washington, D. C., was introduced.

The ladies visited various points of interest about the reservation. The program was brought to a close when the entire group gathered on the parade grounds and watched the cadets in their evening drill. All departed much pleased with the day's entertainment and loud in their praises of the arrangements made by Major Curley, Dr. C. E. DeCamp and others of the local committee.

J. G. WILLS, *Secretary.*

MICHIGAN-OHIO VETERINARY MEDICAL ASSOCIATION

The spring meeting of the Michigan-Ohio Veterinary Medical Association was held on a farm at Blissfield, Mich., May 9, 1935, with more than 50 veterinarians from the two states in attendance. This clinic-meeting was a decided success because there was something of interest doing all of the time, and because it was held in the kind of atmosphere that is familiar to every veterinarian.

Dr. Harry A. Hoopes, of La Rue, Ohio, was right at home at the clinic. He gave a helping hand with all of the clinical material and proved himself to be a specialist in diseases of sheep.

Dr. J. H. Boyd, of Clayton, Mich., was adept with the knife in the castration of a colt and, in addition, gave a splendid demonstration of nerve-blocking before a dehorning operation. Dr. O. W. Schubel, of Quincy, Mich., assisted by Dr. Frank M. McConnell, of Litchfield, Mich., demonstrated a method of casting and restraining cattle for the dehorning operation, using a saw that he has devised for that purpose. The saw is constructed to allow the ears to extend between the back and the blade, thus preventing interference with the operation.

Dr. C. W. Witty, of Elmore, Ohio, operated on a cow for hygroma. Although the operation appeared simple, there were few veterinarians present who had seen it performed. Dr. J. H. Lenfestey, of Lyons, Ohio, Dr. G. L. Noble, of Fayette, Ohio, Dr. H. E. Ash, of Bowling Green, Ohio, Dr. S. G. Colby, of Monroe, Mich., and Dr. A. J. Kline, of Wauseon, Ohio, assisted in making the clinic the splendid success it was. There is no doubt but that such clinics will do more to cement the interests of veterinarians than any other type of meeting.

Dr. E. T. Hallman, president of the Michigan State Veterinary Medical Association, attended the meeting and extended an invitation to all veterinarians to attend the summer meeting of that association.

E. C. W. SCHUBEL, *Secretary.*

MISSISSIPPI VALLEY VETERINARY MEDICAL ASSOCIATION

The third annual all-day clinic of the Mississippi Valley Veterinary Medical Association was held at the fairgrounds, Peoria, Ill., May 23, 1935. Several hundred veterinarians were in attendance from Illinois and nearby states, including Wisconsin, Iowa, Nebraska, Missouri and Indiana.

Dr. A. T. Peters, of Peoria, was general chairman of the committee in charge of the clinic. Assisting him were Drs. W. F. Dixon, H. L. Deuell and W. B. Van Cleave, of Peoria; Dr. E. L. Blumenshine, of Washington, and Dr. R. R. Claybaugh, of Brimfield. The clinic was divided into a number of sections, with Dr. J. W. Lucas, of Abingdon, in charge of the horse section; Dr. W. O. Connell, of Trivoli, in charge of the cattle section; Dr. C. J. Buehler, of Morton, in charge of the sheep section; Dr. J. R. Christian, of Woodhull, in charge of the poultry section; Dr. E. O. Smith, of Princeville, in charge of the swine section, and Dr. Earl R. Kennedy, of Moline, in charge of the

small-animal section. Dr. L. A. Merillat, of Chicago, was in charge of the surgical division, and Dr. R. M. Carter, of Alexis, was in charge of the medical division, with Dr. L. H. Morin, of Clinton, in charge of the restraint division.

Splendid facilities for holding a large clinical demonstration are available in the pavilion of the Peoria fairgrounds, and the Association is the fortunate owner of a large-animal operating table as well as a set of stocks and other equipment. As far as is known, there is not another veterinary association, local or state, in the country that owns equipment of this kind.

The officers of the Association are: President, Dr. F. C. Jones, Macomb; vice-president, Dr. R. M. Carter, Alexis; secretary, Dr. L. A. Gray, Bushnell. Local newspapers gave the clinic some very nice publicity, emphasizing the fact that all operations were performed under either local or general anesthesia.

BUREAU TRANSFERS

Dr. W. R. Kidwell (O. S. U. '19), from Butte, Mont., to Sioux Falls, S. Dak., in charge of meat inspection.

Dr. E. F. Jameson (K. C. V. C. '06), from Detroit, Mich., to Zanesville, Ohio, on meat inspection.

Dr. C. A. Hulbush (McK. '08), from Spokane, Wash., to Walla Walla, Wash., on meat inspection.

Dr. Curtis E. Hagler (Colo. '32), from Topeka, Kan., to Gunnison, Colo., on meat inspection.

Dr. Charles Beverly (Cin. '06), from Chicago, Ill., to Fostoria, Ohio, on virus-serum inspection.

Dr. R. S. Griffin (Chi. '18), from Walla Walla, Wash., to Yakima, Wash., on meat inspection.

Dr. William C. Herrold (O. S. U. '07), from South Omaha, Neb., to Washington, D. C., as assistant chief of the Meat Inspection Division.

Dr. George W. Famous (U. P. '08), from Sioux Falls, S. Dak., to Cincinnati, Ohio, in charge of meat inspection.

Dr. David S. Kay (San Fran. '11), from Seattle, Wash., to South Saint Paul, Minn., on meat inspection.

Dr. Francesco S. Graziadei (Corn. '29), from Buffalo, N. Y., to Chicago, Ill., on meat inspection.

Dr. George E. Aidman (O. S. U. '23), from Indianapolis, Ind., to Richmond, Ind., in charge of meat inspection.

Dr. Henry R. Schrumpf (Ind. '23), from Richmond, Ind., to Indianapolis, Ind., on meat inspection.

Dr. Leo M. Marshall (Iowa '19), from Philadelphia, Pa., to Butte, Mont., in charge of meat inspection.

Dr. Augustus S. Martin (K. C. V. C. '17), from Honolulu, T. H., to Seattle, Wash., in charge of meat inspection.

Dr. Albert H. Julien (O. S. U. '10), from Yakima, Wash., to Honolulu, T. H., on meat inspection.

Dr. Henderson E. Galbreath, Jr. (A. P. I. '25), from Austin, Minn., to Phoenix, Ariz., on meat inspection.

NECROLOGY

A. J. TREMAN

Dr. A. J. Treman, of Wall Lake, Iowa, died at his home on March 22, 1935, following a brief illness brought on by a heart attack. He was 65 years old.

Born at Marshalltown, Iowa, Dr. Treman spent his early life on a farm near Storm Lake, Iowa. He followed farming until 1902, when he decided to study veterinary medicine and entered Iowa State College for that purpose. He was graduated in 1905 and began the practice of his profession at Lake City, Iowa. He remained at Lake City until 1924 when he discontinued his practice for two years because of poor health. In 1926, he located at Wall Lake and resumed practice.

Dr. Treman was active in civic affairs and had served as mayor of Lake City for two terms. Surviving are his widow, a daughter and one brother, Dr. H. B. Treman (McK. '01), of Rockwell City, Iowa. Dr. C. E. Treman (Iowa '30), of Rockwell City, is a nephew, and Dr. P. E. Treman (Iowa '19), of Odebolt, Iowa, a cousin.

WILLARD D. GILCHRIST

Dr. Willard D. Gilchrist, of Jefferson, Iowa, died at the Iowa Methodist Hospital, April 5, 1935, following an illness of several weeks. He was testing cattle in eastern Iowa when he was taken ill and had to be removed to the hospital. Death was due to heart disease and complications.

Born in Boone County, Iowa, August 18, 1879, Dr. Gilchrist studied agriculture for two years at Iowa State College. He then transferred to the Division of Veterinary Medicine and was graduated in 1905. On September 10, 1905, he entered the service of the U. S. Bureau of Animal Industry and was engaged in tuberculosis-eradication work in Iowa. He resigned from the service, March 20, 1919. He later practiced at Williamsburg and Grinnell, Iowa. He held a temporary appointment as U. S. B. A. I. junior veterinarian, with headquarters at Des Moines, from July 6, 1934, until he was stricken on March 18, 1935.

Dr. Gilchrist joined the A. V. M. A. in 1918. He was a member of the Iowa Veterinary Medical Association. Surviving are his widow, four daughters, one son, a twin brother and three sisters.

SAMUEL ECCLES YOUNG

Dr. Samuel E. Young, of Pittsburgh, Pa., died July 17, 1934. Born in Pittsburgh, July 16, 1887, he received his preliminary education in local grade and high schools. He matriculated at the Carnegie Institute of Technology and received the B. S. degree from that institution. Later, Dr. Young entered Ohio State University for the study of veterinary medicine and was graduated in 1917, following which he located for practice in Pittsburgh. He joined the A. V. M. A. in 1919.

FREDERICK JOHN RUFFNER

Dr. Frederick J. Ruffner, of Beloit, Kan., died in a hospital at Halstead, Kan., December 21, 1934, following a brief illness. Following his graduation from high school, Dr. Ruffner entered Kansas State College for the study of veterinary medicine. He completed all courses in the veterinary curriculum, but was held ineligible for his degree, in 1912, because he was lacking half a unit of geometry which the entrance requirements at Kansas demanded at that time. He remained in summer school to make up the required credit, and left school with the understanding that he would receive his degree. When he failed to receive his diploma, he found that his instructor in geometry had not passed him in the course. Dr. Ruffner never returned to college, but he was licensed to practice in Kansas.

R. R. D.

LOUIS E. CHAPMAN

Dr. L. E. Chapman, of Perrysburg, Ohio, died at his home, April 2, 1935, following a heart attack.

Born in New York State, November 20, 1875, Dr. Chapman was a graduate of the Grand Rapids Veterinary College, class of 1904. Following his graduation, he located at Perrysburg, where he practiced until his death. He was a member of Phoenix Masonic Lodge. Surviving are his widow (née Gertrude Salsbery), one son, two daughters and one sister.

ROBERT ERNEST BAKER

Dr. R. E. Baker, of Morristown, Tenn., died in a hospital at Abingdon, Va., April 6, 1935, following an illness with pneumonia. He was 47 years old.

Dr. Baker attended the public schools of Wythe County, Va., and the Virginia Polytechnic Institute, at Blacksburg, before entering the United States College of Veterinary Surgeons, from which he was graduated in 1914. Following his graduation, he practiced for a year at Roanoke, Va., and then removed to Morristown, where he continued in practice until his death.

Dr. Baker joined the A. V. M. A. in 1915. He served as president of the Tennessee Veterinary Medical Association for 1934, and had been secretary of the East Tennessee Veterinary Medical Society for a number of years prior to his death. He was active in civic and church affairs, being a member of the board of stewards of the First M. E. Church, South, and also a member of the choir. Surviving Dr. Baker are his widow (née Mae Cole), an adopted daughter, his parents, four brothers and two sisters.

JOHN T. CHAWK

Dr. John T. Chawk, of Louisville, Ky., died at his home, April 18, 1935, of heart disease. He was 57 years old.

Dr. Chawk was a graduate of Saint Xavier's College, Louisville, and of the Chicago Veterinary College, class of 1904. For the past sixteen years, he had been paddock judge for the American Turf Association, and had served at race meetings at Churchill Downs, Latonia, Lexington and Coney Island. He formerly owned a small stable and raced on the Kentucky and Western circuits. He was a member of the Knights of Columbus, the Catholic Order of Foresters, the Ancient Order of Hibernians and the Mose Green Club. Dr. Chawk is survived by his widow (née Nellie Fineghan) and one brother.

BURT C. McCLINTOCK

Dr. Burt C. McClintock, of Imlay City, Mich., died on April 12, 1935, as the result of injuries received when he was struck by a hit-and-run driver a short time before, near Romeo, Mich.

Dr. McClintock was a native of Meadville, Pa. He was graduated from the Chicago Veterinary College in 1894. He practiced in California until about three years ago, when he located in Michigan. He practiced for a short time at Vicksburg, moved

from there to Schoolcraft, and later settled at Imlay City. Dr. McClintock was licensed to practice veterinary medicine in Pennsylvania, Michigan and Indiana.

CHARLES ROTH

Dr. Charles Roth, of Plainwell, Mich., was instantly killed, March 27, 1935, when he drove his automobile onto a crossing north of Plainwell and collided with a southbound Lake Shore train. The automobile was demolished.

Dr. Roth, who was 55 years old, spent the early years of his life in Caledonia, Mich. He was graduated from the Grand Rapids Veterinary College in 1914, locating shortly thereafter in Plainwell where he had since continued in practice. Surviving are his widow (née Florence Miller), one daughter, one son, two sisters and one brother. Burial was made at Grand Rapids, Mich.

CHARLES H. MARTIN

Dr. Charles H. Martin, of Bronx, N. Y., died on April 14, 1935, following an operation at the Harkness Pavilion of the Columbia-Presbyterian Medical Center, New York City. He was 69 years old.

A native of Dobbs Ferry, N. Y., Dr. Martin was graduated from the American Veterinary College in 1893. During the first 35 years of his career, he lived in Harlem, New York City, where he was well known both as a veterinarian and as a horseman. As veterinarian for the old Lion Brewery, at 107th Street and Columbus Avenue, he attracted wide attention for his success in combating an epidemic of colic, which claimed the lives of 10 per cent of the horses belonging to the brewery. Dr. Martin was the first veterinarian in the city to install summer showers for horses to cool them off during the hot weather. The brewery steeds were given showers every morning and afternoon, reducing the yearly death toll from 40 to four. He served the brewery as chief veterinarian from 1900 to 1920, during which time he showed his prize horse, "Dicker," which was the New York State trotting champion for 1906 and 1907. "Dicker" won 15 loving cups during the 24 years of his lifetime with Dr. Martin, winning a first prize when he was 20 years old. Dr. Martin never acquired another horse after "Dicker" died in 1927.

Dr. Martin removed to the Bronx in 1918, and during the past 15 years had conducted a small-animal hospital at 2400 Jerome

Avenue, where he also had his office. Surviving are his widow and one brother.

R. S. M.

GEORGE E. FETTER

Dr. George E. Fetter, of Hopewell, N. J., died on April 22, 1935, following an illness of four months.

Born in Stockton, N. J., March 7, 1874, Dr. Fetter was educated in the Stockton schools and at the Model School, at Trenton, N. J. He entered the National Veterinary College, Washington, D. C., and was graduated in 1895. The following year, in 1896, he was graduated from the Harvard Veterinary Medical School. That same year he located in Hopewell and practiced there until 1903, when he entered the service of the U. S. Bureau of Animal Industry as an inspector. After serving in this office for about 18 years, he resumed practice in Hopewell.

Dr. Fetter joined the A. V. M. A. in 1918. He was a member of the Veterinary Medical Association of New Jersey, and was active as a member of the First Presbyterian Church of Hopewell, the F. and A. M. Lodge No. 155 and the Hopewell Athletic Club. Surviving are his widow, one son, five sisters and one brother.

J. G. H.

CLYDE R. WALTER

Dr. Clyde R. Walter, of Tulsa, Okla., died at his summer camp at Spavinaw Lake, Okla., April 24, 1935, following an illness of several months with heart disease. Dr. Walter had been confined to his home and unable to attend to the duties of his practice since last October.

Born at Bremen, Ind., April 19, 1880, Dr. Walter received his preliminary education in the public schools of Bremen, and at the Culver Military Academy. Following his graduation from Culver, he entered the Kansas City Veterinary College and was awarded his degree in veterinary medicine in 1908. He located immediately in Tulsa, where he built up a flourishing practice in which he was active until his illness.

Dr. Walter joined the A. V. M. A. in 1922, and was resident secretary for Oklahoma, 1926-1931, during which period he helped to increase the A. V. M. A. membership in Oklahoma just 33⅓ per cent. One of the oldest members of the Oklahoma Veterinary Medical Association, he served as its president in

1925 and held many other important offices in the Association both before and since that time. He was also a member of the Twelfth International Veterinary Congress. The veterinary profession of Oklahoma has lost one of its foremost members and friends, and the city of Tulsa has lost a public-spirited citizen who will long be remembered for his generosity and the untiring interest which he displayed in all civic activities. He was a member of the Presbyterian Church, the Tulsa Rotary Club and the Chamber of Commerce, and was active and prominent in Masonic circles. Surviving Dr. Walter are his widow (née Anna Schroeder) and one sister.

C. H. F.

WILLIAM F. TAYLOR

Dr. William F. Taylor, of Aberdeen, S. Dak., died April 27, 1935. Born in Waukesha Township, Mich., September 3, 1879, he received his early education in local grade schools and in the Athens (Mich.) High School. Following his graduation from high school, he continued with a short preparatory course at Ypsilanti State Normal College and taught for a time. When his father died, in 1902, Dr. Taylor returned to the home farm, which he worked until 1916. In that year, he entered the Grand Rapids Veterinary College and was graduated in 1918. He was a member of the meat-inspection force of the U. S. Bureau of Animal Industry from August 26, 1918, until June 3, 1920, when he resigned. During that time, he was stationed in Chicago. Dr. Taylor received a temporary reappointment in the U. S. B. A. I., September 18, 1934, and was stationed at Sioux City, Iowa. This appointment expired January 31, 1935. He is survived by one half-brother. Interment was at Athens, Mich.

WILLIAM McLAUGHLIN

Dr. William McLaughlin, of Rush City, Minn., died at Saint Andrews Hospital in Minneapolis, April 30, 1935. Death was caused by adenocarcinoma of the stomach with extensive metastasis. Born in Paisley, Ont., November 16, 1870, Dr. McLaughlin removed to North Dakota when he was 18 years old. He lived there for a time and later settled in Minnesota where he spent the remainder of his life. He was graduated from the Chicago Veterinary College in 1912.

Dr. McLaughlin joined the A. V. M. A. in 1916. He was a regular attendant at the meetings of the Minnesota State Vet-

erinary Medical Society and served one term as president. He was a member of Jasper Lodge No. 164, A. F. & A. M., of Rush City Chapter No. 87, R. A. M., and of Jasper Chapter, Eastern Star. To this work he gave much of his time and counsel. At the time of his death, he was a member of the Board of Education of Rush City, and chairman of the Board of Trustees of the Presbyterian Church of that city. Surviving Dr. McLaughlin are his widow (née Margaret Ann Nichol), one son and one daughter.

H. C. H. K.

JOHN NICK LOES

Dr. John N. Loes, of Alburnett, Iowa, died at his home, April 30, 1935, following a long illness.

Born at Garry Owen, Iowa, March 29, 1892, Dr. Loes was educated in the public schools of Dubuque, Iowa, and at the McKillip Veterinary College, from which he was graduated in 1917. He enlisted in the Veterinary Corps and was commissioned a second lieutenant, October 30, 1918. He was ordered to active duty and directed to report at Camp Greenleaf, Ga., for a course of instruction. Illness overtook him and he was sent to General Hospital 14, at Fort Oglethorpe, Ga. Later he was transferred to the Base Hospital, at Camp Dodge, Iowa, and from there to Base Hospital 12, at Biltmore, N. C., where he received his discharge papers, July 26, 1919.

Following his return to Iowa, Dr. Loes practiced for a time at Dubuque, later locating at Alburnett. He was a member of Saint Catherine's Church, of Dubuque, and of Marion Post No. 298 of the American Legion. Surviving Dr. Loes are his widow (née Ruby Clarup) and one daughter.

G. B. M.

FREEMAN PINNEY

Dr. Freeman Pinney, of Chicago, Ill., died at his home, May 1, 1935. He had not been ill, and the news of his death came as a shock to his friends and co-workers.

Born in Wabash County, Ind., January 3, 1886, Dr. Pinney received his early education in the local schools and then attended a commercial school. He decided upon the study of veterinary medicine and entered the Chicago Veterinary College, receiving his degree in 1910. He was appointed to the meat-inspection force of the U. S. Bureau of Animal Industry, in 1930,

and was stationed at New York. The following year he was transferred to Chicago.

Dr. Pinney joined the A. V. M. A. in 1933. He is survived by his widow (née Isel Mae Brown), two sons, his mother, one brother and one sister.

L. G. STICKNEY

Dr. L. G. Stickney, of Toppenish, Wash., died at his home, May 4, 1935, after an illness of eight months. Death was due to a heart ailment.

Born in Canada, June 13, 1882, Dr. Stickney came to the United States when a young man and settled in Wisconsin. He was graduated from the Northwestern School of Pharmacy. Later, he entered the Chicago Veterinary College and was graduated in 1905. In 1912, Dr. Stickney removed to Portland, Ore., where he practiced pharmacy and was City Meat and Milk Inspector. In 1916, he left Portland to start a veterinary practice in Toppenish, where he remained until 1923. In that year, he returned to Portland and stayed there for two years, returning at the end of that time to Toppenish to resume his practice.

Dr. Stickney was active in civic and church affairs. He was head of several committees of the Chamber of Commerce. Surviving are his widow and one daughter. Burial was made in Portland.

R. J. D.

MAJOR EARL F. LONG

Major Earl F. Long, V. C., U. S. A., died at Fort Mills, Philippine Islands, May 7, 1935, following a stroke of paralysis.

Born at Kenton, Ohio, May 8, 1886, Major Long was a graduate of Ohio State University, class of 1910. Following his graduation, he located for practice at Kenton, where he remained for several years. He then joined the forces of the Ohio Bureau of Live Stock Industry and was engaged in regulatory work until the outbreak of the World War. He was commissioned a second lieutenant in the Veterinary Corps, Ohio National Guard, August 5, 1917, and accompanied the 135th Field Artillery, 37th Division, oversea, sailing from New York on June 28, 1918. He served with the 135th and 136th Field Artillery, the 9th Machine Gun Battalion, 3rd Division, and the Wengerohr and Kripp Remount Depots, returning to the United States, November 11, 1919. He was then assigned to the 2nd Division, Camp Travis, Texas, for

duty and was discharged on September 9, 1920, to accept a commission as second lieutenant in the Veterinary Corps, Regular Army. He was advanced to the rank of major on August 5, 1931.

Major Long served at various stations in the United States. Following his graduation from the Army Veterinary School and the Medical Field Service School, in 1925, he served as instructor for the R. O. T. C. Veterinary Unit, Ohio State University, from July 7, 1925 to August 11, 1931. His last assignment was at Fort Mills, where he served as Station Veterinarian.

Major Long joined the A. V. M. A. in 1925. He was affiliated also with the Masonic Fraternity, the Knights of Pythias, the Maccabees and the Eagles. Burial was made in Arlington Cemetery, Washington, D. C. Surviving are his widow and one sister.

D. M. S.

WILLIAM EDWARD MULDOON

Dr. William E. Muldoon, of Peru, Ind., died at his home, May 12, 1935, following a heart attack.

Born in Truxton, N. Y., November 4, 1888, Dr. Muldoon attended local schools and the West Virginia Military Academy before entering the New York State Veterinary College at Cornell University. Following his graduation, in 1913, Dr. Muldoon remained at the College as an instructor in the Department of Materia Medica, serving in that position until 1918. He also took postgraduate work and was granted an A. M. degree in 1916. Dr. Muldoon was made an associate professor in 1918 and held that title until 1919, although at that time he was officially in the service of the United States Army. He was commissioned a second lieutenant on November 19, 1917, and ordered to report to Camp Greenleaf, Ga., for a course of instruction. On April 8, 1918, he was assigned to the Veterinary Training School, at Camp Lee, Va., as an instructor, and was made a first lieutenant on May 30, 1918. He was elevated to a captaincy on July 14, 1918, and was discharged from the service, May 3, 1919. He was commissioned a major in the Organized Reserve, September 16, 1923.

In 1919, a short time after his discharge from military service, Dr. Muldoon joined the veterinary faculty of Kansas State College as associate professor of medicine. He became a full professor a year later. In 1923, he returned to his home in Waverly, N. Y., where he remained for a time. He then removed to Evanston, Ill., where he was associated with Dr. J. V. Lacroix for about four months. He left Evanston to locate in Peru, Ind., for gen-

eral practice. While in Peru he was chief veterinarian to Cole Brothers Circus. He had officiated as referee in a number of Big Ten Conference football games.

Dr. Muldoon joined the A. V. M. A. in 1918. He served as resident secretary for Kansas, 1920-22, and was the A. V. M. A. representative to the Horse Association of America, 1922-23. He was secretary (1920-21) and chairman (1921-22) of the Section on General Practice. He is survived by his widow (née Blanche Horner), his mother, and several brothers and sisters.

HENRY CHARLES BABCOCK

Dr. Henry C. Babcock, of Miami, Fla., died in a hospital, May 26, 1935. He was found shot through the temple on the grounds of his "millionaires' row" estate. Near him was found a note which read: "Cause of this act—health gone—feel that my mind is going; farewell and love to all."

Born in Masonville, Iowa, August 11, 1873, Dr. Babcock was graduated from the Ontario Veterinary College with the class of 1894. He then entered the Louisville Medical College and received the M. D. degree in 1896. Later, he became a member of the faculty and taught materia medica at the Kansas City Veterinary College. During this time, he continued his veterinary studies at the Kansas City Veterinary College and was awarded the degree, Doctor of Veterinary Science, in 1904. He remained on the faculty until 1905. In the meantime he received an appointment as inspector with the U. S. Bureau of Animal Industry and was stationed at Kansas City, Kan. He held this position until April 1, 1909, when he established an office for the practice of human medicine in Kansas City, Mo. At the same time, he was active with further courses in medicine. He was granted degrees from the Kansas City Hahnemann Medical College in 1913; from the Eclectic Medical College, Kansas City, Mo., in 1914, and from the Kansas City College of Medicine and Surgery, in 1916. Dr. Babcock was licensed to practice medicine in Florida in 1914, and built up a lucrative practice in Miami. He was a member and a fellow of the American Medical Association.

Our sympathy goes out to Dr. Earl F. Graves, of Madison, Wis., in the death of his wife, February 17, 1935, after a long illness; and to Dr. Charles H. Higgins, of New York, N. Y., in the death of his wife recently.

PERSONALS

Dr. C. H. Betty (Mich. '26) has resumed general practice at Mattoon, Ill.

Dr. H. E. Wicker (Iowa '32) has located for general practice at Stratford, Wis.

Dr. E. M. Dobbs (K. S. C. '16) has changed locations from Oakland to San Leandro, Calif.

Dr. P. E. Johnson (Chi. '14) has requested a change of address from Stockton to Modesto, Calif.

Dr. Edwin R. Perrin (Geo. Wash. '12), of Northville, Mich., has located for practice in Imlay City, Mich.

Dr. G. A. Ledgerwood (Ont. '14), of Valcartier Camp, Quebec, Canada, has removed to Palmerston, Ont.

Dr. Peter C. Molgard (K. C. V. C. '14), formerly of Eldora, Iowa, is now located in Charles City, same state.

Dr. C. C. Sundstrom (Colo. '30), formerly of South Gate, Calif., has removed to Manhattan Beach, same state.

Dr. H. S. Van Vranken (K. C. V. C. '10) is now established at Story City, Iowa, removing there from Burt, Iowa.

Dr. Martin L. Hutchins (U. P. '16) has requested a change of address from Sunbury, Pa., to Rock Glen, same state.

Dr. Ralph R. Cusack (Chi. '17), formerly of Carrington, N. Dak., has located for practice at Jamestown, N. Dak.

Dr. John W. Van Vliet (K. S. C. '22) has removed from Holton, Kan., to Jefferson City, Mo. Address: 308 Brooks Street.

Dr. William R. Kermen (Wash. '34), who has been in Los Angeles, Calif., for the past year, has returned to Cle Elum, Wash.

Dr. Milton R. Fisher (O. S. U. '25) has been appointed supervisor of milk control in the Health Department of Saint Louis, Mo.

Dr. A. C. Merrick (O. S. U. '24) has resigned from the U. S. Bureau of Animal Industry and entered general practice at Brookfield, Ill.

Dr. Alex Copland, Jr. (Mich. '24), of Grosse Pointe Farms, Mich., has been appointed veterinarian for the Detroit Racing Association.

Dr. J. Lynn Leonard (Corn. '09), of Astoria, L. I., was a hospital patient recently, this being his sixth hospital experience in three years.

Dr. Cecil Moulton (San Fran. '18), who was doing relief work for the State up to April 1, is now back on meat inspection at Modesto, Calif.

Dr. C. E. Mummert (Ind. '07), of Logansport, Ind., has reopened his office following his recovery from an illness which incapacitated him for several weeks.

Dr. G. D. Pinder (Ont. '34) is now associated with Dr. R. G. Cuthbert (Ont. '23) in operating the Fourth Avenue Veterinary Hospital, Vancouver, B. C.

Dr. J. D. Nolan (Wash. '32), formerly of Raymond, Wash., has requested that his Journal be sent to him at 1202 College Ave., East Saint Louis, Ill.

Dr. B. F. Davis (K. C. V. C. '07) is secretary of the Colorado Stock Growers' and Feeders' Association, with headquarters in the Keeley Building, Denver, Colo.

Dr. H. D. Bergman (Iowa '10), of Iowa State College, is serving as president of the Missouri Valley Intercollegiate Athletic Association (Big Six Conference).

DR. JOHN B. KOERNER (Gr. Rap. '11), of Sykesville, Md., was recently elected mayor of that city. Dr. Koerner has been a member of the City Council for several years.

DR. H. D. PORT (Chi. '14), State Veterinarian of Wyoming, Cheyenne, recently came through a long period of convalescence, following an attack of influenza aggravated by complications.

DR. GEORGE G. FERLING (Ont. '83) and Mrs. Ferling, of Richmond, Ind., celebrated their golden wedding anniversary recently. Dr. Ferling has practiced in Richmond for more than 50 years.

DR. N. L. SIPLOCK (Ont. '31) has resigned his position with the Department of Health, of Cleveland, Ohio, to enter practice in that city. He is connected with the Cleveland Small-Animal Hospital.

DR. CLIFFORD C. WAGNER (O. S. U. '26), of Cleveland, Ohio, is busy with his practice again following his return from the Marine Hospital, Cleveland, where he was confined for two months with pneumonia.

DR. B. SCOTT FRITZ (U. P. '17) has resigned his position with the Pennsylvania Bureau of Animal Industry, effective May 15, and has accepted a position with the Gilliland Laboratories, at Marietta, Pa.

DR. ALEXANDER GOW, JR. (Corn. '29), of College Park, Md., has been granted a year's leave of absence from the Live Stock Sanitary Service Laboratory, and has located for general practice at Frederick, Md.

DR. DAVID H. BACHTEL (O. S. U. '33), of Canton, Ohio, is building a new small-animal hospital at the corner of Cleveland Avenue and 33rd Street, N. W., which he expects to occupy about the middle of June.

DR. F. O. CONOVER (Chi. '04), of Petersburg, Ill., was the victim of an accident, on April 22, which left him with a broken leg. Dr. Conover was attending a colt, when the animal fell on him and crushed his leg.

DR. WILLIAM BELL (K. C. V. C. '92), of Nashville, Tenn., has been named to the Tennessee State Board of Veterinary Medical Examiners by Governor McAlister, to succeed Dr. George R. White (Col. '97), of Nashville.

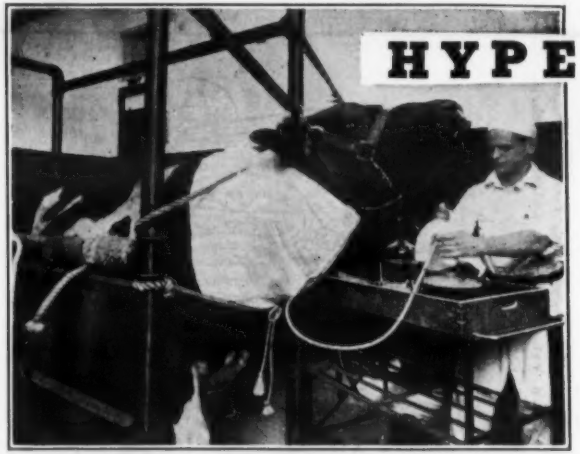
DR. G. H. CALDWELL (Chi. '15), of Wheaton, Ill., was seriously injured on May 8, when his automobile struck a culvert, near Belvidere, Ill. The impact drove the headrail of the culvert through the car, breaking both of Dr. Caldwell's jaws and fracturing his skull.

DR. J. H. MOORE (Chi. '15), who has been associated in practice with Dr. Benjamin McInnes (R. C. V. S. '74) and his son, Dr. B. Kater McInnes (U. P. '11), at 57 Queen Street, Charleston, S. C., has opened an office for general practice at 60 Queen Street.

DR. J. H. FIEGE (Mich. '25), of Kenosha, Wis., is building a new small-animal hospital at 3804 Sixty-third Street, to take the place of his present frame structure which will be torn down. The new hospital, which will cost about $3,000, will be of concrete and tile construction.

DR. J. C. STOKES (Cin. '12), of Calumet City, Ind., a member of the veterinary staff of the Western Weighing and Inspection Bureau, addressed the Kiwanis Club, of Hammond, Ind., on May 23. His address covered the activities of the present-day veterinarian, with particular emphasis on food inspection.

DR. CARL J. FOX (Mich. '26), formerly of Standish, Mich., has returned to Michigan after a six-year stay in California and has opened a hospital for large and small animals at Bancroft. Dr. Fox served as assistant state veterinarian of California for four years, and for two years was a dairy superintendent and veterinarian on a California ranch.



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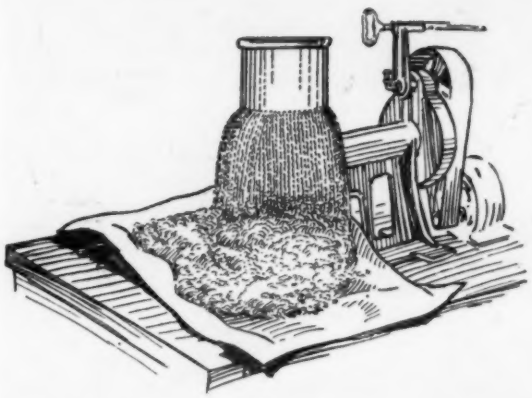

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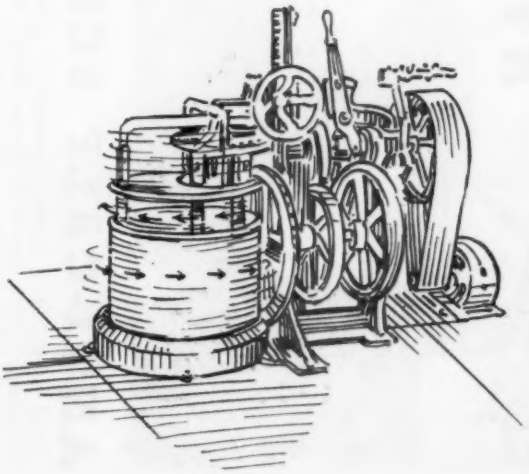

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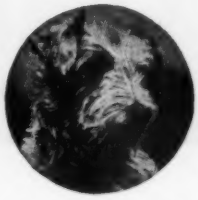

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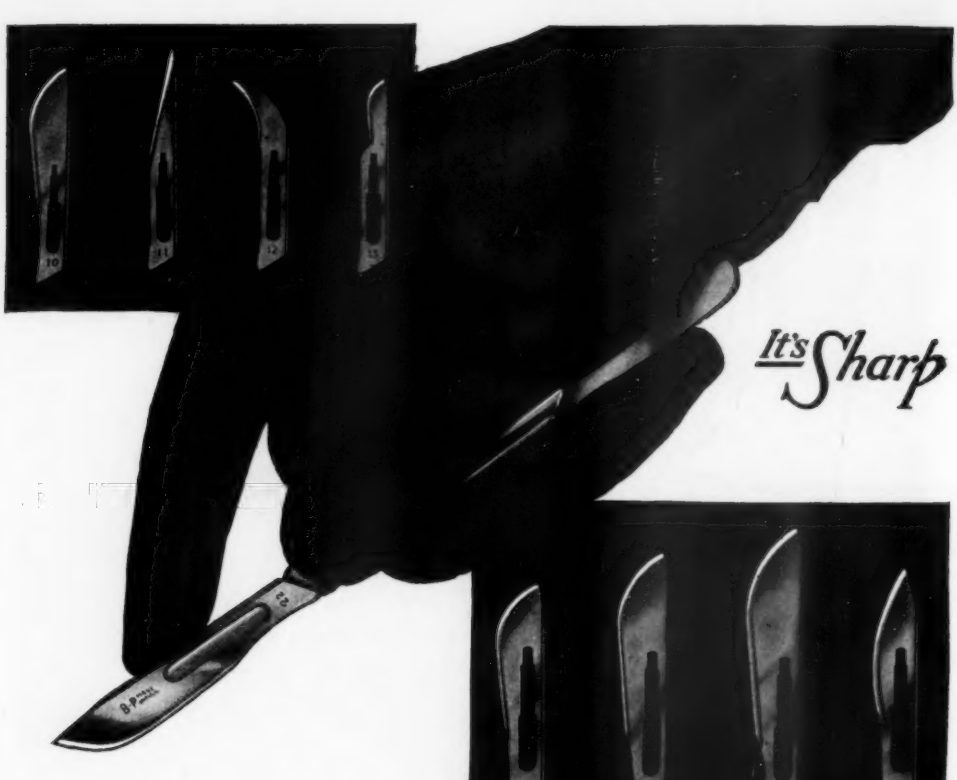

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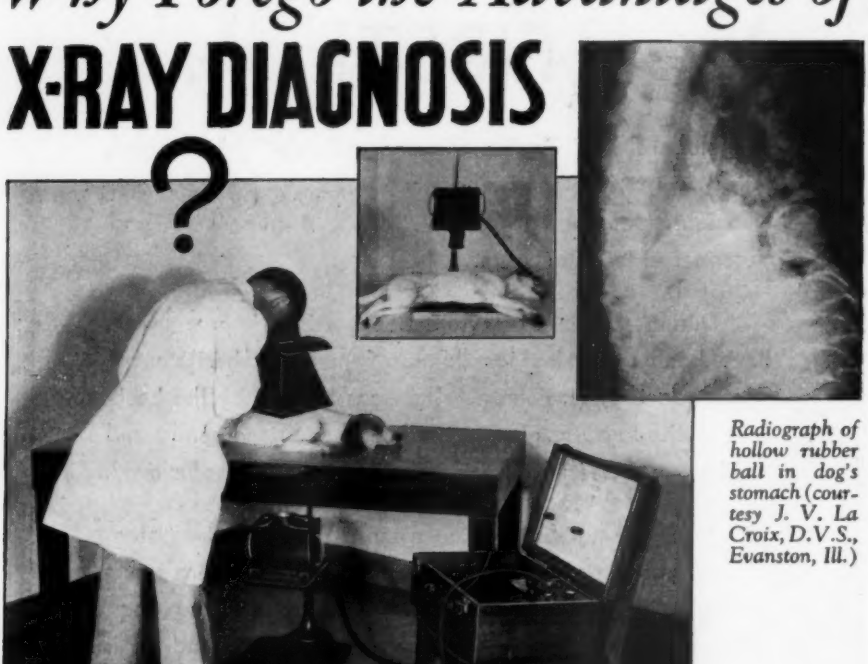

Radiograph of hollow rubber ball in dog's stomach (courtesy J. V. La Croix, D.V.S., Evanston, Ill.)

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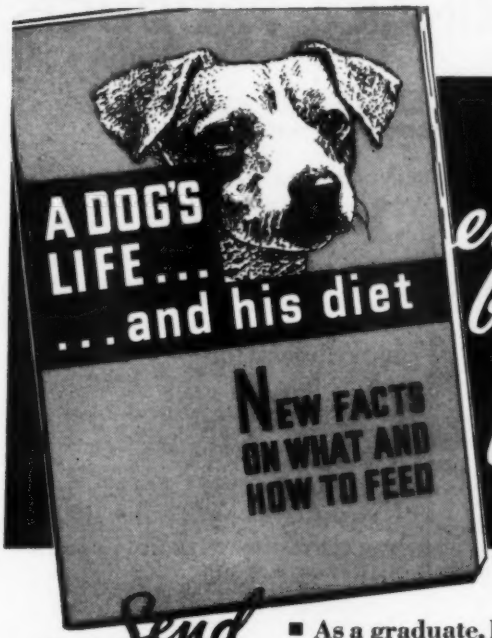

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ANTACID TABLETS

Seven grains; for the correction of acidosis and gastro-intestinal hyperacidity in dogs and cats.

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Supplied enteric coated or uncoated. 1/8, 1/4, 1/2 grain tablets.

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2½ grains (0.162 grams) each drug; antipyretic and intestinal antiseptic for small animals.

Dose: One tablet 3 or 4 times daily.

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BARBITAN (Barbital-Acetanilid)

Hypnotic, analgesic and antipyretic. For the relief of nervous manifestations, pain and fever associated with diseases and trauma of small animals.

Dose: One tablet every 3 hours.

No. 1—Barbital, 1½ gr.
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No. 2—Barbital, 4 gr.
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Packages: 50 and 1000 tablets of each size.

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A Cholagogue Laxative.

Dose: One or 2 tablets daily for dogs, cats, foxes, etc., as required.

No. 1—Purified Bile Salts, 1 gr.
Ext. Cascara, ½ gr.
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Packages: 100 and 500 tablets.

BLACK TONGUE IN DOGS

See Hepatic Extract

CALCIUM GLUCONATE TABLETS

Five grains: for skin and chronic infections.

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Packages: 100 and 1000 (5 gr.).

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A rapid cardiac and central nervous system stimulant.

Dose: Small animals 5-10 cc.; horses and cattle, 10 to 30 cc. intramuscularly.

Package: Six 10 cc. ampuls.

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A cardiovascular stimulant for shock.

Packages: 1 oz. vial; 12-1 cc. ampuls (sterile); 5 cc. vial (sterile).

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A specific for Black Tongue and Anemia in dogs.

Dose: Intramuscular injections of 5 cc.

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A conditioning food for dogs, cats, foxes, etc.

Packages: Twelve 8-oz. tins.

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1/10 grain each gland.

Dosage: One tablet 3 times daily.

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Packages: 10 cc. vial and 6—1 cc. vials.

SALINE SOLUTION (RINGERS SALTS) TABLETS—Twelve grains.

Packages: 100 and 1000 12-grain tablets.

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Arrest Hemorrhage.

Packages: 20 cc. vials for local or hypodermic use.

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Chemically pure tetrachlorethylene and anhydrous magnesium sulphate.

For the removal of roundworms and hookworms in animals.

Size **AA**— .2 cc. (3 minims)
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Size **C**—2.6 cc. (40 minims)

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Size **1**—0.3 grams.
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Packages: 50, 100 and 250 capsules.

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Equine Encephalomyelitis Vaccine (Cutter), prepared after the method of Shahan and Giltner of the B. A. I., is now available.

Although still in the experimental field, this vaccine gives promise of being the most satisfactory method of controlling encephalomyelitis. It has been proven safe from the standpoint of administration, and produces an active immunity in from two to three weeks. Two 25-cc doses are given at intervals of seven to fourteen days, starting two to four weeks ahead of the time that the disease has appeared in previous years.

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NRW—250 cc Western Type Serum.....$6.00
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Vaccine

NQW—1 immunization (2 25-cc doses) Western Type.$2.50
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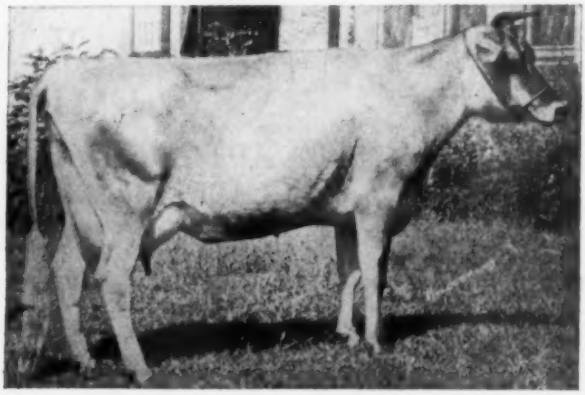
Iodigen is a quick-acting compound containing 80% of elemental iodine, that has replaced potassium iodide because of the latter's failings.

The toxic reactions of KI (rash, lacrimation, paralysis) appear before the alterative action of its iodine can be exerted. On that account its use has been largely discarded in both human and veterinary medicine.



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Junior FORT DODGE
BIO-CHEMIC REVIEW

JUNE 1935

CAMERA!

CIRCUS TIME'S HERE — And the big show mixes its pulchritude and pachyderms. Photo © Keystone.

CHIMP ADOPTS PUP — In Los Angeles this 3-year-old simian is mothering fox terrier pup. © Keystone.

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Wheat Germ Oil for Functional Sterility

• With "More milk—more butter fat!" the demand of the dairy industry, bovine rations have been arranged primarily to meet that demand, heedless of the increasing deficiency of those diets in the substances essential to normal conception and reproduction.

The effective therapeutic value of Fort Dodge Wheat Germ Oil in treating functional sterility has been proved in the nearly two years this product has been on the market. In addition to increasing spermatogenesis and stimulating ovulation, it is also indicated in any condition — in all species—where there is a lack of Vitamin E or A.

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STORY Of-The-Month

• Son had just heard a new word. "Father," he asked, "what do you mean by business ethics?"

"Well," answered the Old Man, "it's this way. The other day a customer came into the store and bought a pair of $10 shoes. On the way to the cash register I noticed that there were two $10 bills stuck together. Now here is where business ethics come in . . . should I tell my partner?"

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Suitable for use intramuscularly and on the feed

FOR TREATMENT OF FUNCTIONAL STERILITY

in all species—by increasing spermatogenesis (development of sperm cells in tubules) of males, and by stimulating ovulation of females. . . .

Suggestions for handling

for STALLIONS, JACKS, BULLS, etc.

1. Make 25% of ration freshly rolled or crushed wheat during breeding season.
2. Supplement with 1 teaspoonful Jen-Sal Wheat Germ Oil, cold pressed, on feed twice daily, for stallions, jacks, bulls. May be supplemented further by weekly intramuscular injections of 10 to 20 cc.
3. Exercise stallions, jacks and bulls daily to promote all glandular activity—important as a gonad stimulant. Jen-Sal Kay-I Tabs (20 gr. Potassium Iodide) also may be employed.

for MARES, COWS, etc.

For 30 days prior to breeding, feed freshly crushed or rolled wheat, with teaspoonful doses Jen-Sal Wheat Germ Oil, cold pressed, twice daily. Inject weekly, 10 to 20 cc Wheat Germ Oil intramuscularly. Kay-I tabs may be used as alterative to stimulate glandular activity. Inject 10 cc Jen-Sal Oestrual Hormone just prior to oestrum.

* * *

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WHEAT GERM OIL,
Cold Pressed

250 cc
(30-day treatment)

$4.50

20 cc 60c

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